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DEPARTMENT OF THE INTERIOR

UNITED STATES BUREAU OF MINES
JOHN W. FINCH, DIRECTOR

INFORMATION CIRCULAR

REVIEW OF LITERATURE ON EFFECTS OF BREATHING DUSTS WITH SPECIAL REFERENCE TO SILICOSIS

PART I

CHAPTER 1. DEFINITION AND CLASSIFICATION OF DUSTS

CHAPTER 2. SOURCES OF EXPOSURE TO DUSTS

CHAPTER 3. PHYSIOLOGICAL EFFECTS OF BREATHING DUSTS

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BY

D. HARRINGTON AND SARA J. DAVENPORT

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DEPARTMENT OF THE INTERIOR - BUREAU OF MINES

REVIEW OF LITERATURE ON EFFECTS OF BREATHING DUSTS, WITH SPECIAL REFERENCE TO SILICOSIS[1]

PART I

By D. Harrington[2] and Sara J. Davenport[3]

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INTRODUCTION

This circular presents part I of a review of the literature on effects of breathing dusts, with special reference to silicosis, largely in the mining and allied industries. The literature on this subject is so extensive that no attempt has been made to include all published material but only to assemble some of the more outstanding data on several aspects of the subject. Parts II and III will deal with prevention and treatment of dust diseases and some of the economic and legal aspects of dust diseases in industry, with special reference to mining.

Dust diseases are much before the public at present, and there is good reason to believe that the information on the subject available to the layman is extremely meager; even those rated as experts usually have a more or less theoretical conception of the diseases, their causation, remedies, and prevention, rather than actual experience with them. Until recently little attention has been given to prevention, and the usual attitude of the engineer, upon whom the main reliance for prevention ultimately must devolve, has been to let "sleeping dogs lie" or, more definitely, to "let well enough alone." However, the "laissez faire" attitude no longer is tenable, and in the past few years numerous conditions have caused those engaged in industry, including mining, to bring the subject before the public with a view to ascertaining the facts upon which to formulate remedial measures, primarily preventive.

CHAPTER 1. DEFINITION AND CLASSIFICATION OF DUSTS

According to Webster's Unabridged Dictionary dust may be defined as fine, dry particles of earth or other matter so comminuted that they may be raised and wafted by the wind; that which is crumbled to minute portions; fine powder. In 1876 Richardson (1)[4] included in the term "dusts" all those fine and solid particles thrown off from various substances in the processes of manufacture or treatment of articles in common use in daily life. Drinker (2) in 1930 defined dusts as solid particles ranging in size from less than 1 micron[5] to about 150 microns; this definition is probably as applicable as can be had insofar as concerns respiration.

Physiologically, there are almost as many different classifications of dust as there are authors on the subject. Richardson (1) suggested the following: (a) Cutting dusts, formed of minute, hard, crystallized particles with sharp, cutting, and pointed edges and composed of iron or steel, stone, sand or glass, dried silicates in earthenware, lime, and pearl; (b) irritant dusts derived from woods, ivory, textile fabrics, fluffs of wool, silk, cotton, flax, hemp, hair, and clay; (c) inorganic poisonous dusts charged with arsenical salts, derived from poisonous chemical compounds used for coloring artistic products or for preserving organic substances, such as furs; (d) soluble saline dusts, organic poisonous dusts thrown off during the making of tobacco into cigars and snuff and carrying with them particles of the dried tobacco plant; (e) obstructive and irritating dusts composed of carbon, fine particles of coal dust, scrapings of carbon or soot, dust of rouge, and flour.

Baskerville (3) in 1912 classified dusts as: (a) Insoluble inorganic dusts, including metals (antimony, arsenic, type metal, brass, bronze, copper, aluminum, iron, steel, lead, manganese, vanadium and ferrovanadium, silver, tin, zinc, and solder) in a state of fine division (dusts, atomized metals, metallic powders); flue dusts; various ore dusts (iron ore); silica; sand, emery, flint, glass powders; carbon graphite, diamond, coal, soot; brick dust, marble, granite, cement, terra cotta; lime, gypsum, plaster, meerschaum; phosphates; and guano. (b) Soluble inorganic dusts, including substances likely to be swallowed and absorbed, such as metal particles, including lead, brass, copper, zinc, arsenic, mercury, and silver, as well as soluble inorganic salts. (c) Organic dusts, comprising such widely varying materials as sawdust, fur, skins, feathers, broom straw, grains, flours, jute, flax, hemp, cotton, wool, carpet dust, street sweepings, tobacco-box dust, hides and leather, felts, rags, paper, and horsehair.

In 1918 Hoffman (4) considered the following classification to be in strict accord with the facts as they were known and understood at that time:

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[4] Numbers in parentheses refer to bibliography at end of circular.

[5] A micron is 1/1,000 of a millimeter or approximately 1/25,000 of an inch.

(a) Inorganic dusts, including metallic dust, mineral dust, and dusts of the mineral industries; (b) organic and miscellaneous dusts, including vegetable-fiber dust, animal and mixed-fiber dust, organic dust, and mixed organic and inorganic (public) dusts.

Thompson (5) classified dusts as (a) insoluble inorganic dusts (irritating the respiratory passages), as flint, silica, sand, carbon (coal, soot), brick dust, marble, granite, terra cotta, cement, asphalt, enamel, glass, quartz, lime (gypsum, plaster), meerschaum, phosphate (fertilizers), guano, emery, diamond dust, metal filings (lead, brass, iron, steel, etc.), pumice, and ashes; (b) soluble inorganic dusts (liable to be swallowed and absorbed), as soluble arsenic, mercury, lead and silver compounds, metal filings of lead, brass, and zinc; (c) organic dusts and fibers arising from handling or manufacture of wood, bone, and shell, from fur, skins, hides and leather, feathers, brooms and straw, flour and grain, jute, flax (linen), hemp, cotton, wool (worsted, etc.), tobacco, felt, carpets, rags and paper, horsehair, and street sweepings.

Heim de Balsac and Agasse-Lafont (6) suggested the following classification: (a) Active dusts, which are immediately disseminated and radiate beyond their point of application; toxic dusts (lead, arsenic, and mercury); caustic dusts, chromates, etc.; infectious dusts. (b) Inert dusts, soft flexible felting dusts (wool, cotton, and leathers); hard, troublesome, wounding dusts (ligneous, metallic, stone, and coal dusts).

Schürmann (7) differentiated dust, according to origin, into animal, plant, and mineral dust and dust from the artifacts. Animal dust is evolved with the working of ivory, horn, whalebone, bones, mother-of-pearl, hides, leather, bristles, sheep's wool, hair (horse, rabbit, and cow), and feathers; plant dust has its origin in industries working with grain, medical powders, spices, cotton, hemp, jute, flax, tobacco, wood, thick-shelled nuts, bark of plants (tanning industry), rags, shoddy, and paper; mineral dust is formed in the working of hard coal, marble, and other limestones and in connection with clay and porcelain industries, also pumice, sandstone, granite, and slates; dust from artifacts is found in the glass, glazing, enameling, tile, cement, Thomas slag, celluloid, iron, steel, bronze, galalith, and lead-alloy industries and in the chemical (especially dye) industry. He defined mixed dust as a mixture of polishing materials and the fragments of the object being polished.

According to Drinker (2) all these classifications are without much practical importance, since in daily practice in industry the harmful effect is exercised more frequently by dusts mostly of mixed origin. Besides, what is most important is the physical and chemical constitution of the dusts and consequently their action on the system. An effort has been made to group under the heading "inert" a certain number of dusts; the number, however, is becoming more and more reduced since certain dusts, such as talc and asbestos, until recently considered as inert nevertheless have caused serious organic lesions among certain classes of workers. The majority of experts admit that the so-called "inert" dusts are not really inert when considered from the pathogenic point of view. Although at the outset they are not harmful and even when present in the workroom in large quantities some of them cause only transitory discomfort (sneezing, watering of the eyes, cough), it is certain that as the organic reactions diminish and the system seems to have recovered from the effects, they nevertheless finally attack it in a subtle manner, sometimes even seriously. The mucous membrane of the nose, the conjunctiva, and first the upper and subsequently the lower respiratory passages, the teeth, the skin, and often the digestive tract are subjected to the harmful action of these dusts.

CHAPTER 2. SOURCES OF EXPOSURE TO DUST

The prehistoric man who originated the trade of making stone implements probably started the first industrial hazard, the extent and severity of which the medical profession, as well as the industries concerned, has begun to realize only recently. In fact, dust as a factor in the causation of disease had not received much attention before 1900. At the site of one of the Swiss lake dwellings where flint implements were found, although the flint must have come from a distance (probably from the South of France) the chippings of material were in such profusion as to imply that the implements were manufactured on the spot. In other words, a prehistoric flint-knapping factory was probably located there (8). Some of the persons who prepared these implements no doubt suffered from respiratory diseases. According to Collis (9) the flint-knappers of Brandon, the lineal occupational representatives of this oldest of industries, who still use tools similar in shape to the deerhorn picks of their prehistoric ancestors, suffer a terrible mortality from phthisis induced by flint dust generated in their work. The work is carried on in small workshops at the back of cottages in the rural district bordering Norfolk and Suffolk, where large flints or potstones are found; the district has been known from remote antiquity for the manufacture of arrowheads and other prehistoric implements, tinder boxes, and in later years flints for flintlock guns.

Dusts of various kinds are carried in the atmosphere in all parts of the world, and the inhalation of these dusts over periods of years inevitably produce changes in the lungs, as for example the pigmented lung of the city dweller (10). Another example of the general exposure to dusts is given in the report by Soper (11), published in 1906, on the air and dust in the 21-mile subway of the Interborough Rapid Transit Railroad of New York City. Chemical analysis of the dust underground showed that it contained 61.30 percent of iron, nearly all of which was in the metallic state, 21.94 percent of organic matter of vegetable and animal origin, 15.58 percent of silica and other matters insoluble in acid, and 1.18 percent of oil. There were on the average 61.6 mg of dust in 1,000 cubic feet of air; the maximum weight was 204 mg. The dust in the air of the subway was 11 to 800 percent heavier than that in the street air. Although some of this dust was carried in from the streets, much of it was of underground origin from the gradual wear and tear of the wood, cement, and other materials used in construction of the subway and also from the operation of the trains, for the largest percentage was iron dust from the grinding action of the powerful brakes on the cars. The loss of weight in the brake-shoes alone was estimated as about 1 ton per mile of track per month, and when the loss from the wheels and the railway track is added to this the origin of the metallic dust is explained. The men had not been employed underground long enough at that time to show the effect on the health of inhalation of the dust. Although no serious disease was found among them many suffered from inflammatory affections of the nose, throat, and windpipe and also from "dry pleurisy" unaccompanied by pain. More than 20,000 workmen are said to be engaged in dusty occupations in New York City alone (11).

Hoffman (4) prepared an occupational grouping to emphasize in a general way the principal dust hazards in 118 occupations or groups of employment, which he said was in strict accord with the facts as they were known and understood at that time (1918). Under the heading "organic and miscellaneous dusts" he listed 65 occupations; under the heading "inorganic dusts" he listed 52 occupations.

He gave the number of persons employed in the dusty trades, according to the United States census for 1910, as 3,928,978; of these 1,667,181 were listed as employed in occupations exposed to metallic and mineral dust and dust in the mineral industries. He considered that quantitatively the most important kind of metallic dust, as met under typical industrial conditions, is the dust of iron and steel, which, however, is generally more or less intermixed with dust of other metallic or mineral substances. Mineral dust exposure is most common in the stone industry, among potters, in cement manufacture, and in mining.

In a paper on the extent and severity of the health hazard from dust in the mines and allied industries in the United States van Siclen (12) gives the following estimates, based on the 1929 decennial Federal Census, of the number of men exposed to dust hazard in various types of mining and industries working in metals and minerals:

	Total exposed
Metal mining	62,268
Nonmetallics mining, omitting tunnel and foundation excavations	23,665
Bituminous-coal mining, underground	450,513
Bituminous-coal mining, open pit	8,219
Anthracite mining, underground	143,063
Anthracite mining, independent washeries	269
Smelting, nonferrous plants	13,166
Smelting, ferrous plants	24,960
Cement plants	33,368
Abrasive industry	3,873
Asbestos products	8,092
Clay products	93,336
Cutlery	14,991
Glass manufacture	67,527
Granite, slate, marble, and other stone products	28,715
Hones, whetstones, and similar products	174
Iron and steel	39,697
Mineral fertilizers	20,926
Minerals and earths, ground	1,679
Nonferrous metal alloys and products	79,183
Pottery, including porcelainware	33,409
Sand-lime brick	566

In his conclusion he states:

> No totaling of the number of workmen exposed to the dust hazard in the mining and allied industries in the United States, as estimated in the preceding pages, has been undertaken; first, because the estimated figures themselves are subject to revision after more detailed study of the mineral industries and manufactures, and second, because the degree of the dust hazard varies with the several industries, with the manifold occupations in each industry, and between similar positions in different plants of the same industry. A lump-sum total would therefore have no real meaning. The separate totals, however, are sufficient to indicate the wide extent of this kind of hazard.

In Great Britain in 1930 some 35,000 workers, excluding coal miners, were employed in the refractories industry, tin mining, pottery industry, metal grinding, sandstone-working, sand-blasting, and other trades in which dust is a prominent feature (10). In 1931, 877,141 men were employed in the coal mines of Great Britain. In 1930, 287,540 men were employed in the mines of France, 155,397 in the mines of Belgium, and 400,654 in the mines of Prussia (excluding lignite mines) (13). About 300,000 men are employed in the mines of South Africa (14). Probably 150,000 persons are employed in the quarrying, cement, and pottery and stoneware industries of Germany (15).

The above figures on number of employees in the dusty trades are given merely to indicate the possible exposure to the dust hazard and, of course, are in no way complete figures of the actual number subjected to the hazard. According to Lanza (16) the prevalence of silicosis has been estimated carefully in a few instances, but the rates so obtained cannot be applied generally because industrial conditions vary so widely. However, it is apparent that silicosis is a widespread industrial hazard, is probably increasing, and affects appreciably the death rate among industrial workers exposed.

Only dusts encountered in the mining and allied industries and the diseases caused by them will be considered in this paper.

CHAPTER 3. PHYSIOLOGICAL EFFECTS OF BREATHING DUST

Historical Résumé

Probably the oldest published data regarding the harmfulness of exposure to dust is the statement in Pliny's Natural History (17) that -

> Minimum refiners in the factory envelop their faces with loose bladders, which enable them to see without inhaling the fatal dust.

Hippocrates (18), who was born about 460 B.C., called attention to the difficult breathing of the metal digger. Celsus, a Roman medical writer who lived in the first century, states:

> By far the most terrible form of emaciation is that which the Greeks call phthisis. It spreads to the lungs. On top of this ulceration occurs and a slow fever which at times disappears and at other times reappears.

In quoting the above Mavrogordato (19) says that one may assume that when early writers discussed dust phthisis the disease they had in mind was of the nature mentioned by Celsus.

In 1551 Amatus Lusitanus (7) reported that most workers occupied with the preparation of gypsum and lime died of lung phthisis.

Agricola (20) in his De Re Metallica, published in 1556, described mining as "a perilous occupation to pursue because the miners are sometimes killed by the pestilential air which they breathe; sometimes their lungs rot away."

He also states (20):

> Some mines are very dry and the constant dust enters the blood and lungs, producing the difficulty of breathing the Greeks call asthma. When the dust is corrosive it ulcerates the lungs and produces consumption; hence it is that in the Carpathian Mountains there are women who have married seven husbands, all of whom this dreadful disease has brought to an early grave.

According to Mavrogordato (19) we learn from Georgius Agricola that in the sixteenth century it was known that permanent injury to the lungs resulted from exposure to certain kinds of dust but not to all kinds. Two kinds of injurious dusts were acknowledged - a noncorrosive and a corrosive type. Each of these classes was associated with a different kind of lung change - "simple" and "infective" silicosis, respectively, described in the 1916 General Report of the Miners' Phthisis Prevention Committee of South Africa.

Paracelsus (21), a Swiss alchemist and physician, in his book on miners' phthisis and other miners' diseases described especially the chronic lung troubles of miners as "lung consumption", "asthma", and "dyspnea." He was the first to list briefly the occupational diseases of miners and smelters as well as the first in world literature to prepare a monograph on industrial medicine (21). From 1531 to 1534 Paracelsus revisited the mines and smelters of Schwaz in the Tyrol where he had worked as a laborer between 1510 and 1520 and wrote his book, which was published in 1567 after his death. He found that -

> Miners in metal mines, when they are occupied in digging, smelting, and washing gold, silver, salt, alum, sulphur, lead, copper, zinc, iron, and quicksilver in the refining of vitriol, suffer from various disturbances of the lungs, stomach, and intestines; they are then said to have "miners' diseases." However, there is nothing found in the old writers in regard to these diseases.

His theory was that lung diseases contracted aboveground depended upon climatic conditions, which were influenced by the rays of the stars. Similarly, pulmonary disease of miners underground was caused by mineral rays. However, he philosophically reminded his readers that -

> We need metals and, therefore, we must risk life and health for them, since everywhere in nature good and evil lie together. As the crocodile distresses and kills men by his breath, likewise also the vapors (fine dust?) of such metals kill us. * * *. The organism must be prevented from coming in contact with the metal emanations; for if the organism is once injured there is no cure.

A present-day author expressed a similar idea when he said (22) that the increasing interest in the subject is a recognition of the condition of pneumoconiosis as a more or less necessary risk of commercial development in the progress of civilization in recent years.

It is said that the early death of Paracelsus was due to injuries to health received during his activities in the mining and metallurgical industries.

According to Schürmann (7), Pansa, in 1614, was the first to discuss in detail lung diseases of miners and the asthma of grain measurers. In 1649 Diemerbroek (23) is said to have made the first section of a stonecutter's lung which, in a case of fatal asthma, revealed "lung vesicles completely clogged with fine dust"; he is quoted by Ramazzini (24) as finding such heaps of sand that in running the knife through the pulmonary vesicles he thought he was cutting some sandy body.

In 1652 Ursinus (25) wrote of the lung disease that attacked especially the miner but also the smelter. He divided lung diseases into two classes - one was caused by poison, and the other was not. The principal symptoms he described were coughing and shortness of breath. Etiology was a question of breathing dust, catching cold, fatigue, injury, and the like. He said the lung disease due to "poison" was not so clear and was unknown before Paracelsus.

In his book published in 1656 Stockhausen (26) defined miners phthisis as "difficult breathing, chest asthma with disagreeable hard cough and considerable hoarseness, which effects generally degenerate into fatal consumption!" He attributed these symptoms to metallic vapors, dust, and meteorological conditions, from which miners suffer more or less and call "miners' consumption" because the affliction arises especially in connection with mining. The actual cause he attributed to "especially thick, lowering clouds and mine dampness, watery humidity, all sorts of vapors of earth and metal, various fumes and smoke, different dusts and dirt." He also said that much dust arose from grinding so that workers were forced to cover nose and mouth with cloths.

In 1690 Löhneiss (27), referring to miners, gave the morbid sequence of events as follows:

> The dust and stones fall upon the lungs, the men have lung disease, breathe with difficulty, and at last take consumption.

Ramazzini (7) was the first to recognize the social significance of industrial diseases from chemical substances. In his book published in 1700 he mentioned the harmful effect of dust on the respiratory organs. For information he went to the workers in the occupations, entered the mines, ascended the mine shafts, and collected the experiences of his occupational colleagues. His book was based on a study lasting more than 40 years.

Referring to the fact that flint dust had long been recognized in England as injurious Collis (28) said that a patent for grinding flints by a wet method had been granted in 1713 to Thomas Benson, of Newcastle-under-Lyme; previous to this time flints were pounded dry,

> which process proved very destructive to mankind insomuch that any person, ever so healthful and strong, working in that business, cannot possibly survive over 2 years, occasioned by the dust sucked into his body by the air he breathes.

In 1783 Hoffman (29) stated that breathing metal fumes and vapors, as well as dust, caused the disease picture of "miners' phthisis."

According to Henkel (30) (1745) the external causes or occasion of miners' phthisis were dust from stones and metals, lack of air, bad air, or bad fumes or vapors.

In 1770 Scheffler (31) wrote that the description of miners' phthisis given by previous medical men was insufficient. His conception of it was a

> chronic slow fever with hardening of the glands and obstruction of the lungs with a lack of elaboration, secretion, nutrition, and apposition which through the arsenical or other dusts dries out the soft lymph of the glands in the lungs and air passages.

The symptoms given by him were slight fever, loss of appetite, cough, shortness of breath, and swollen feet, the patients finally becoming bedfast and showing great weakness, irregular pulse, sleeplessness, dry skin, usually constant fever, often bloody sputum, hemorrhage, sweating, and diarrhea until death claimed them.

In 1780 Ackerman (32) called miners' phthisis without doubt the most "frequent and specific" mineworkers' disease and said that physicians did not agree regarding the true definition of this disease.

As the result of a special inquiry into the prevalence of dust phthisis Allison (33) found that rarely did a mason, regularly employed in hewing stones in Edinburgh, live free from phthisical symptoms to the age of 50. According to Mavrogordato (20), Allison, a professor of medicine at Edinburgh, established the association between dust phthisis and tuberculosis, that is, between dust phthisis and true phthisis. It was not dust per se that was responsible for the "ulceratio" of Celsus but a secondary complication. The corrosive dust of sixteenth-century Agricola and eighteenth-century Ramazzini became the "tuberculosis" of Allison. Allison like Laennec knew of the anatomical tubercle but not of the tubercle bacillus.

Hugh Miller (34), the famous writer, geologist, and stonemason in telling of his own narrow escape wrote:

> The dust of the stone which I had been hewing for the last 2 years had begun to affect my lungs, as they had been affected in the last autumn of my apprenticeship, but much more severely; and I was too palpably sinking in flesh and strength to render it safe for me to encounter the consequences of another season of hard work as a stonecutter. From the stage of the malady at which I had already arrived, poor workmen, unable to do what I did, throw themselves loose from their employment, and sink in 6 or 8 months into the grave - some at an earlier, some at a later period of life; but, so general is the affection that few of our Edinburgh stonecutters pass their fortieth year unscathed, and not 1 out of every 50 of their number ever reaches his forty-fifth year.

Thackrah (35), however, claimed that such generalization was not justified and stated that "dust of every kind irritates but not in an equal degree." He recognized that masons inhaling particles of sand and dust which arise from chipping stone were short-lived, generally dying before they attained the age of 40. Thackrah also dwelt on the prevalence of phthisis among the metal grinders of Sheffield and quoted Knight's opinion that fork grinding ought to be confined to criminals. A few years later Holland (36) portrayed the conditions of fork grinders -

a picture of wretchedness which has no parallel in the annals of any country, or in the records of any trade. Fiction can add no color or touches to a picture like this. Truth transcends the gaudy embellishments of imagination. The distempered fancy has here no room to exercise her powers.

Incidence of Dust Diseases

According to Greenburg (37) data on prevalence of occupational disease in different countries and at different periods of time must be interpreted with the greatest caution, because industrial processes differ so widely and change so frequently. To be conclusive such data should be available in the form of actual death rates, based on knowledge of the population exposed as well as on the number of deaths occurring, and properly corrected for the age distribution of the group involved.

A brief summary of available information on the incidence of dust diseases in some of the principal countries of the world follows.

Great Britain

In 1862 Royal Commissioners were appointed in England (9) to inquire into the health of men employed in metalliferous mines. Notwithstanding the evidence of several witnesses, particularly the miners themselves, that dust was far worse than anything else with which they had to contend, the commissioners concluded on practically unanimous medical opinion that the influence of dust was subsidiary to the many other adverse conditions of ventilation, exposure to fumes of explosives, and variations of temperature which at that time were prevalent in the mining industry. Collis (9) said this conclusion was unfortunate, as the prevalence of phthisis in certain industries was attributed to imperfect hygienic conditions rather than to dust inhalation, a point then in dispute.

In 1902 a departmental committee (9), of which Haldane was a member, was appointed to reinvestigate the cause of the still persistently high phthisis mortality among Cornish tin miners. This committee decided that -

> So far as the Cornish miners are concerned it seems evident enough that stone dust which they inhale produces permanent injury of the lungs - gradually in the case of ordinary miners, and rapidly in the case of machine drillmen - and that this injury, while it is apparently capable of gradually producing by itself great impairment of the respiratory functions, and indirectly of the general health, also predisposes enormously to tuberculosis of the lungs, so that a large proportion of miners die from tubercular phthisis. That the primary injury to the lungs is due solely to inhalation of stone dust would seem to be practically certain.

From February 1, 1919, when the first scheme of compensation for silicosis was made for the refractories industry, until the end of 1928 compensation awards were made in England in 423 cases, including 121 deaths

(38). During the period 1921-23, 328 deaths were recorded for England and Wales under the generic title "chronic interstitial pneumonia", which included such diseases as fibroid phthisis, fibrosis of the lungs, silicosis, and miners' phthisis, when returned as nontuberculous (39). In 1926 the question was asked in Parliament (40) as to the number of men not affected by the Silicosis Act but when examined by officers of the Home Office during the preceding 5 years had been found suffering from the disease. The reply was that no routine examination of workers had been made by the medical inspectors of factories except sample examinations in connection with special inquiries into the grinding and other industries. Of 1,106 workers employed in processes involving silica dust so examined 556 were found to be affected by fibrosis of the lungs; 528 of these men were employed in the grinding industries and 28 at steelworks.

A memorandum on industrial silicosis and asbestosis issued by the Home Office in July 1932 (41) stated:

> The incidence of silicosis continues to be serious and widespread, as is shown by the following figures of cases in which compensation has been paid under the special schemes made under section 47 of the Workmen's Compensation Act, 1925. During the last 3 years there have been 80 cases, including 30 deaths, amongst workmen employed in ganister mines and silica brickworks; 179 cases, including 25 deaths, in the getting and manipulation of sandstone at quarries or on premises worked in conjunction therewith; 322 cases, including 87 deaths, in the pottery industry; 81 cases, including 32 deaths, in the metal industries, including metal grinding and sandblasting; and 91 cases, including 20 deaths, in coal mines.

According to Stewart (42) in districts of Lancashire County, where the industry of coal mining predominates (40 percent of the occupied males being engaged in mining), the male death rate from pulmonary tuberculosis is higher than the corresponding rate for the whole administrative county.

Sladden (43) stated that during the 4 years 1925-28 he had examined the bodies of 20 coal miners; 1 had advanced silicosis and 7 other slighter degrees of the condition. Since 1929, 63 examinations were made, 27 of which showed definite and serious degrees of silicosis and 16 others slighter degrees. In this connection Haldane (43) said he preferred the more general term "pneumoconiosis" for these cases. Among anthracite miners he found the deaths of 30 of 83 bodies examined definitely attributable to pneumoconiosis. He stated that in all cases, whether anthracite or nonanthracite, a high percentage of silica was the outstanding feature in chemical analyses of the lungs.

The 1930 report of the chief inspector of factories and workshops (44) states:

> By a recent arrangement the factory-inspection service receives copies of all death certificates in which death resulted from pulmonary disease involving fibrosis of the lungs. Of 700 such certificates

received in 1930, 241 gave silicosis as the cause of death and in the great majority of cases it was found that the previous occupation of the persons concerned was one in which there was recognized exposure to silica dust.

The industries furnishing the greatest number of such cases were the pottery trade, 52; sandstone, 49; coal mining, 39; sand-blasting, 10; tin mining, 10; and other industries, 90. The report also said that recent investigations of the effects of exposure to asbestos dust had resulted in the adoption of measures to control dust in the textile branch of the asbestos industry. Data regarding 20 fatal cases of asbestosis without tuberculosis show a serious hazard in continued exposure to heavy concentrations of asbestos dust.

In 1931 Wood and Gloyne (45) were prompted by the recent discovery that asbestos workers are often the victims of occupational disease to determine whether pulmonary asbestosis also paves the way for a tuberculous invasion of the lungs or aggravates an existing infection. They studied 57 cases of pulmonary asbestosis, including 18 males and 39 females, 8 of whom were young girls from 18 to 21 years old; 12 of the 57 cases showed evidence of pulmonary tuberculosis, 4 of the 12 died, and on post-mortem examination evidence of obsolescent tuberculosis was found in 2 and of active tuberculosis in 2.

Merewether (46) found 4 active cases of tuberculosis and 33 inactive on examination of 374 asbestos operatives while at work. According to Sparks (47) 2,000 workers in England are exposed to asbestos dust.

An inquiry by Sutherland and Bryson (48) on the occurrence of silicosis among sandstone workers in England since February 1, 1929, under the Workmen's Compensation Act of 1925 disclosed that 1 of every 4 men at work appeared to have "masons' disease." As only men at work were examined cases as far advanced as third stage and many in second stage, not being at work, were not included in the survey.

The outstanding feature of an investigation by Ferguson (49) of the occurrence of silicosis among sandstone workers in Scotland and the North of England was the high degree of pulmonary morbidity existing throughout the industry. Of 1,000 working people examined 433 showed clinical evidence of pulmonary fibrosis. Radiological examination of all the workers showing clinical evidence of fibrosis was impracticable but doubtless would have disclosed other cases of silicosis; most of the cases X-rayed by Ferguson were those showing the severest fibrosis and those which gave the most reason for suspecting the presence of silicosis. One hundred and seventy-three of these workers showing clinical evidence of pulmonary fibrosis were X-rayed; 97 showed first-stage and 12 second-stage silicosis (advanced, nodules coalescent).

The report of the chief inspector of factories and workshops in Great Britain for 1932 (50) considers particularly 42 deaths from asbestosis or asbestosis with tuberculosis and 281 deaths from silicosis or silicosis with tuberculosis distributed among the industries as follows: Pottery, 147; sandstone, 60; grinding materials, 30; sand-blasting, 23; scouring powder, 5; miscellaneous, 16.

Under the heading "Parliamentary Intelligence" the Colliery Guardian (51) on August 4, 1933, published the following:

> In the House of Commons on Friday, on the motion for the summer adjournment, Mr. R. Davies introduced a discussion on the annual report of the Chief Inspector of Factories, in the course of which Mr. Tinker raised a number of questions relating to industrial diseases and the solvency of employers, as a remedy for which he demanded a system of compulsory insurance. Mr. R. T. Evans suggested that the bronchial diseases particularly affecting miners should be placed in a separate category and dealt with by the Mines Department under a new order. Mr. E. Williams complained that scores of men had died from silicosis in South Wales, whose dependents had not received a farthing of compensation and that there were also scores who were dying but were not receiving compensation after having worked for years in hard ground, because the rock did not contain a certain amount of silica. He also urged that anthracosis should be scheduled as an industrial disease.

On February 16, 1934, the Colliery Guardian (52) published a statement from the Home Office that since June 1, 1931, when the coal-mining industry first came within the scope of the General Medical Board under the Silicosis Compensation Scheme, 432 men in the industry had been certified by the board as disabled or having died from silicosis or from silicosis accompanied by tuberculosis and that 385 of these cases occurred in the South Wales coal field. Earlier comparable figures were not available.

On March 16, 1934, The Iron and Coal Trades Review (53) quoted the following statement by Prof. J. S. Haldane from the British Medical Journal:

> Cases of silicosis in the sense normally accepted end nearly always in death from phthisis, and if there were actually a large number of cases of real silicosis in this sense among South Wales colliers, their phthisis death rate would be considerably increased above the normal figure. There seems to be no sound reason for believing that many more cases of real silicosis occur among South Wales colliers than among other colliers, and I think the widespread alarm produced in the South Wales colliery district by the numerous certified cases of silicosis has no substantial basis so far as silicosis is concerned.

However, Harper (54), a radiologist practicing in the South Wales coal field, in a letter to the British Medical Journal published in May 1934 said that when the full facts were known there was little mystery in the many cases of silicosis found in that part of South Wales. Deductions are made from the pay of the coal miners at the colliery offices in behalf of the medical men in the district; therefore the workmen can have roentgenological examinations if the family doctor is doubtful of their condition, and silicosis at present is the fashionable disease in the area. Harper (54) states that it is probably safe to say that more colliery workmen have been X-rayed in this area than in any other part of the coal field; hence more cases of silicosis have been found than in other areas where no such examinations are made, which partly explains why there are more known cases here. In fact, Harper said, there are more cases of silicosis than have been either examined or passed by the board because some refused to be examined, preferring to continue at work regardless of the cost to their health rather than be retired by compulsion with partial compensation of about £1 a week to meet the necessities of life and no further hope of employment. He quoted Kettle as saying that nearly all the specimens sent to the board from this area were those of infective silicosis, and the infecting organism was most commonly the tubercle bacillus. Harper considered this statement much more reliable than any returns of the Registrar General. Therefore there would seem to be no difference between the silicosis in South Wales and the commonly accepted type, and he could find no roentgenological difference between cases of silicosis in South Wales and cases he had seen in the Belgian coal fields.

Germany

In Germany industrial pathology was developed at the beginning of the nineteenth century simultaneously with the upward trend of German industry (7). In 1781 Tissot (55) graphically illustrated the health hazards encountered by stonemasons; he stated that in one place where the people formerly had been engaged in felling trees and in carving they were the "handsomest, strongest, and healthiest of persons" but for 25 years - since the inhabitants had turned to stonecutting - there had been in that region the most lingering diseases. In 1804 and 1812 Goetzinger (56) in describing the mountainous part of Saxony said that the fine dust and drink brought the stonecutter to an early death.

According to Schürmann (7) only a few works have been published on the harmful effects of iron and steel dust to which the metal polisher is exposed, in spite of the fact that the works of Paracelsus, Ramazzini, and others were known in Germany, Ramazzini's work being the sole textbook in the field of occupational disease used in Germany up to the middle of the nineteenth century. In contrast to the English reports on the health picture of the Sheffield polishers, Hauer (57) in 1832 reported conditions among German polishers as not unfavorable to health. Nasse (58) was the first to report in regard to polishers' disease; he wrote that the grinding workers lasted only 12 years and recommended respiratory protection for them. The first known German publication on the harmful effects of dust on polishers was issued by Pappenheim (59) in 1860. In 1866 Zenker (60) determined iron oxide in a lung and called the disease caused thereby "siderosis." He designated the diseases of the lungs caused by dusts as "pneumonoconiosis." In a book by Schlockow (61) on the care of health and medical statistics in Prussian mines, published in 1881, the statement is made that of 942 miners examined by Seltmann 37.7 percent had emphysema of the lungs.

Only 5 roentgenologically normal pictures were found among 25 sand-blasters examined by Lochtkemper (62) in 1930; the remaining 20 (80 percent) revealed various stages of silicosis. Thiele (63) found that 28 percent of porcelain workers examined by him were affected by pneumoconiosis; Holzmann and Harms determined roentgenologically the presence of pneumoconiosis in 75.6 percent of 41 selected porcelain workers; and Koelsch found 58 percent of 19 workers affected. Böhme (64) determined pneumoconiosis in 28 percent of 184 coal cutters who had worked more than 10 years underground and in 66 percent of hard-rock miners; in another investigation he determined by X-ray examination that 36.2 percent of 69 hard-rock miners had pneumoconiosis and by clinical examination 17.3 percent. Komissaruk (65) examined clinically and roentgenologically 40 foundryworkers who had worked in dusty industries for more than 10 years; 4 definitely had pneumoconiosis; 5 were doubtful, and 3 were borderline cases. In 23 cases the X-ray revealed old inactive pulmonary tuberculosis, but no tubercle bacilli were found in the sputum.

In 1932 Lochtkemper and Teleky (66) published the results of an extensive study on dust which covered working conditions and health of workers in many different dusty trades. However, the number of workers examined in each group was usually too small for definite conclusions. Examination of 22 women employed in a scouring-powder factory in 1930 revealed that all but 3 were affected by silicosis in varying degree, 6 being in stage 3. Examination of 7 workers in a sand and cement plant resulted in the diagnosis of 1 case of silicosis in the first stage and 2 in the third; the others showed no indication of silicosis or only slight pneumoconiosis which the authors classify as "silicosis 0-I." Inquiry among workers and physicians of the region revealed that 4 had died and 2 were still ill of tuberculosis. Six of 7 shell limestone cutters examined were diagnosed as having pneumoconiosis 0-I and 1 silicosis 0-I. Of 18 graywacke workers examined 3 were diagnosed as negative, 4 as having silicosis 0-I, 4 silicosis I, 3 silicosis II, 1 silicosis I-II, 1 silicosis III, and 2 silicosis II with tuberculosis.

From an examination of the statistics of a large sick benefit association Koelsch and Kaestle (67) found the illness frequency, especially for tuberculosis and diseases of the respiratory organs, less for the quarry and shell limestone workers than for the other members of the association, but the mortality rates apparently were greater for the stone workers (0.35 percent) than for the others (0.21 percent). These investigators examined 82 persons who had worked in shell limestone only; 14 had more or less definite signs of dust lung, but it differed in appearance from the silicosis of the sandstone worker. They concluded from these examinations that under certain conditions limestone also can cause changes in the lungs within the meaning of dust lung if it can have a correspondingly long action. Dust lung was more frequent and more pronounced among those who had worked in limestone and sandstone, the more so the closer the work in sandstone had followed upon that in shell limestone.

According to Landau (68), the cleaning of metallic castings presents great health hazards to the men engaged in the work. Among those examined 69 percent had pneumoconiosis and 8 percent tuberculosis. Work with steel seemed to be much more dangerous than with cast iron, probably because compressed-air tools were used in cleaning the steel castings which were cast in a mold of sand very rich in silica.

Australia

In 1902 while a system of sewers was being dug in Sydney, Australia, a Sewer Works Ventilation Board (69) was appointed to inquire into the working conditions and to recommend means of improvement whereby the work could be rendered less hazardous. The board found that -

> For many years past miners employed in this class of work (tunnelling) suffered acutely from a disease which was for a long time known by the rather misleading term "sewer disease", but as the complaint became more widely known it was strongly suspected that dust was the chief cause of the mischief.

The board blamed the fumes of explosives and the expired air from the lungs of the miners in imperfectly ventilated tunnels as causes contributory to the high mortality but stated that the dust from hammering, drilling, and the use of the pickax was probably the sole cause of the disease once known as "sewer disease."

In 1912 Armstrong (70) investigated a reported epidemic of pneumonia at Broken Hill and found that the death rate from pneumonia among underground miners in that locality during 1910-12 was 6.5 per 1,000, or nearly 4 times as great as that for all males in New South Wales. In 1914 a Royal Commission was appointed to inquire into the mining industry at Broken Hill. With regard to industrial diseases this commission stated that pneumoconiosis and plumbism were among the risks of a miner's calling, while other diseases - for example, pneumonia - were added risks but not strictly industrial because they affected others than miners. From unanimous medical testimony the commission concluded (70) that pneumonia was more prevalent, severe, and fatal among miners in Broken Hill than among any other class in the State. Although attributing the prime cause to sudden changes in temperature the commission advanced the opinion that dust inhalation in any form would predispose to disease. It also expressed the opinion that pneumoconiosis was virtually unknown at Broken Hill and blamed mining in other States for such cases as had been noted. Tuberculosis, however, was a disease to which the miners at Broken Hill, as elsewhere, were peculiarly subject, and the presence of tuberculous patients in a mine was considered particularly dangerous.

In 1919 the Technical Commission (70), constituted to examine the miners at Broken Hill, for the first time in Australia used the X-ray as a means of diagnosis of silicosis as an occupational disease. Of 4,337 mine employees examined 193 were diagnosed as having pneumoconiosis - 90 in the first stage, 44 in the second, and 59 complicated with tuberculosis; 39 cases were definitely diagnosed as suffering from pulmonary tuberculosis only and 26 others were thought to be suffering from uncomplicated pulmonary tuberculosis. Of 2,618 underground men examined in 1922, 266 showed pneumoconiosis - 113 in the first stage, 51 in the second stage, and 102 complicated by tuberculosis; 107 additional men were diagnosed as suffering from tuberculosis only.

In 1922 the New South Wales Board of Trade, in conjunction with the Commonwealth Department of Health (70), investigated clinically and radiologically the prevalence of pulmonary diseases among workers in sandstone and other siliceous rocks in the metropolitan district of Sydney; of 716 men examined 123 were found to be suffering from silicosis. Using the standards set by the Broken Hill Commission, 47 of these were diagnosed as being in the first stage, 38 in the second, and 38 suffering from pneumoconiosis complicated by tuberculosis; 16 were diagnosed as having pulmonary tuberculosis only.

The Workers' Compensation Commission of New South Wales (71) reported on 1,300 of 1,500 workmen examined up to August 23, 1929, as follows: Of 107 stonemasons examined 9 had tuberculosis, 17 were in the various stages of silicosis, and 26 had silicosis with tuberculosis. These men had worked in the industry from 10 to 55 years. Of 29 monumental masons examined 2 had tuberculosis, 1 class A silicosis, and 5 silicosis with tuberculosis; of 37 ballast quarrymen 1 had tuberculosis and 2 marked silicosis; of 80 dimension quarrymen 6 had tuberculosis, 3 early silicosis, 2 marked silicosis, and 16 silicosis with tuberculosis. Of the 377 rock choppers examined 12 had tuberculosis, 25 silicosis, and 10 silicosis with tuberculosis.

In 1931 Moore (71) reported an investigation in New South Wales to determine the incidence of fibrous pneumoconiosis among coal miners; 471 volunteers who had worked at least 10 years underground were examined by the Division of Industrial Hygiene; 199 came from 1 mine, several employees of which had had pulmonary fibrosis; the remaining 277 volunteers were employed in 8 other mines of the district. The percentage of cases of fibrosis for all men examined was 25.9. The percentage of fibrosis among men with coal-mining history only ranged from 22.5 to 25.7 percent; the percentage incidence among men who had worked also in metal mines or quarries as well as in coal mines was 39.7.

In Tasmania in 1928 (69) the various mining centers were visited in turn and men examined clinically and radiologically virtually at the mine head; 65 percent of the mine employees available were examined. Of 314 underground workers 5.9 percent were suffering from uncomplicated silicosis and 2.1 percent from silicosis with tuberculosis. Of all workers examined 1.1 percent were suffering from tuberculosis only - 1.2 percent of the underground and 1 percent of the surface workers.

In 1910 a Royal Commission appointed to report on pulmonary diseases among the miners in the Western Australian gold-mining industry (69) concluded that -

> The miner is more liable to lung disease generally than the average male over 15 years of age. The miner is less long-lived than the average male over 15, partly on account of greater liability to lung diseases.

> Tuberculosis of the lungs is on the increase among miners and is twice as prevalent as among males over 15.
>
> Pneumonia among the acute, and bronchitis, asthma, emphysema, and fibrosis of the lungs among the chronic lung diseases are more prevalent among miners than among males over 15.

The principle was laid down that -

> Any man suffers from fibrosis to the extent to which he is exposed to the continued inhalation of mineral dust. If there is no dust, there will be no fibrosis, and conversely, the continued inhalation of dust certainly produces fibrosis.

During an investigation (72) in 1925-26 of pulmonary conditions of mine employees in Western Australia 3,039 men were examined at Kalgoorlie and 1,028 at 9 other mining centers throughout the State. Of the 4,067 men examined 798 (19.6 percent) presented definite evidence of pulmonary silicosis and 155 (3.8 percent) had tuberculosis; 12 of the latter group had tuberculosis without silicosis. Of the 1,759 surface workers 6.6 percent had silicosis, 1.5 percent silicosis with tuberculosis, and 0.3 percent tuberculosis alone. The surface workers engaged in dry milling without previous underground experience showed the highest incidence of silicosis - 9.6 percent were affected compared with 2.2 percent of the employees engaged in wet milling without dry-milling experience, and 2.0 percent of those who had had no wet- or dry-milling experience. Of the 2,308 underground workers 23.3 percent had silicosis, 5 percent silicosis with tuberculosis, and 0.3 percent tuberculosis alone. No fewer than 51.5 percent of the workers engaged in developing and 39.2 percent in stoping were affected with silicosis or tuberculosis, in contrast with 8.4 percent of those without stoping or developing experience.

Since 1926 the mine employees in Western Australia have been examined annually by the Commonwealth Department of Health (72). Of 2,290 men previously classed as normal 30 (1.3 percent) were found on reexamination in 1927 to be suffering from uncomplicated silicosis and 13 (0.5 percent) had tuberculosis only; of 491 diagnosed as silicotic 86 (17.5 percent) had silicosis with tuberculosis. In 1928, of 2,822 men classed as normal the previous year, 48 (1.7 percent) had progressed to silicosis only, 11 (0.4 percent) had progressed to silicosis plus tuberculosis, and 3 (0.1 percent) had simple pulmonary tuberculosis; of 425 silicotics 25 (5.9 percent) on reexamination showed signs of tuberculous complication. In 1929, of 2,293 normals reexamined, 100 were diagnosed as silicotic. Silicosis was not found in any case under 40 years of age or with less than 5 years of underground work.

In 1907 a report on miners' phthisis to the Committee of the Bendigo Hospital, Victoria, Australia (69) stated that there was undue mortality among Bendigo miners due to respiratory diseases, notably tuberculosis. The cases examined were classified according to two clinical types - a pure fibrosis of the lungs, nontuberculous in origin, silicosis; and a mixed type with a tuberculous infection in a fibroid lung. Of 61 men examined in Bendigo in 1928, 9.4 percent of the underground workers were diagnosed as suffering from uncomplicated silicosis and 7.5 percent from silicosis and tuberculosis; 3.3 percent of all workers were found to have tuberculosis only.

Netherlands

In the Netherlands (73) a medical examination of 2,013 stonemasons in 1899 revealed 169 tuberculous cases and 10.8 percent strongly suspected of having tuberculosis. During the period 1891-1900 the death rate among stonemasons for all men between 18 and 50 years of age was greater than in any other occupation. Even such injurious occupations as typesetting, cigar-making, and diamond-cutting showed considerably lower death rates. The death rate in the age group 18-24 years differed little from the average for all occupations; the highest death rate was noted between 36 and 50 years. Of 952 stonemasons dying during 1886-98, 87 percent died of diseases of the respiratory organs. The first general examination of adult stonemasons took place in 1923, the second in 1926, and the third in 1929. In 1923 in nearly half of the cases the X-ray examination gave more information than the physical examination.. Of 69 cases in 1929 lung affections were not shown clinically in 19 but were shown in the X-ray examination and in 38 cases both clinically and by X-ray.

As a result of examination of stonemasons who in the course of their lives had worked on certain kinds of stone and others who had worked on more than one kind of stone, Kranenburg (73) reported that of 16 sandstone workers 2 were in the second stage and 5 in the third stage of silicosis after 20 to 30 years' employment in the occupation; of a total of 74 Belgian limestone masons only 5 were in the second stage and none in the third stage after the same number of years' employment in the occupation. Using the third stage as a test, the proportion was more unfavorable among the 90 stonemasons working various kinds of stone including sandstone than among the Belgian limestone workers, although relatively favorable in comparison with the sandstone workers. A striking fact was the large number - 18 - of Belgian limestone workers who showed no affection after 10 to 30 years' employment compared with only 4 stonemasons working on all kinds of stone. Fewer cases of third-stage silicosis were found among Belgian limestone workers after 40 to 60 years' employment or longer than among sandstone workers.

In his general conclusions Kranenburg (73) made the following statement regarding the effects of sandstone and limestone:

> The working of sandstone alone must be regarded as more injurious to the lungs than the working of limestone alone. The working of sandstone alone does not always lead to symptoms of silicosis and, on the other hand, the working of limestone alone (Belgian limestone, marble) does not prevent silicosis in a serious form. The working of sandstone and limestone alternately appears to produce less serious results in the same period than the working of sandstone alone.

Italy

In Italy the first clinical and anatomical observations (74) probably were made by Ramazzini. In 1906 Biondi (75) observed that the greatest damage to the miners in the mines of Sardinia was due to the action of dusts from gangue containing silicates. He also noticed that virtually all coal miners were addicted to coughing and continued to expectorate black sputum even some time after they had quit their occupation. He found minor dust injuries among the Bergamo miners in 1907, perhaps because they labored intermittently in mines and on farms. However, in a few miners, especially the older men, Biondi found cases of pharyngitis and bronchial catarrh, with dark sputum due largely to the black smoke of the candles used, and he observed that acute affections of the respiratory apparatus were more serious and more obstinate in these workers.

In 26 post-mortem examinations of Sadinian miners Frongia (76) in 1908 noted that the cause of death was in most cases an acute infection (pneumonia); he also found among these workers many cases of serious and diffused arteriosclerosis, emphysema, and anthracotic foci, with more or less diffused softening of the tissues and gangrenous foci containing a black brothlike matter - the characteristic finding of black phthisis. On the other hand, he found foci of bronchiectasis due to tuberculous infection in only 5 cases. Eighty-five percent of the fatal cases among these miners therefore were not caused by tuberculous infection.

Pesenti, Rota, and Finzi (77) in 1906 reported on health conditions of workers in lime, cement, and plaster. Pesenti affirmed the high incidence of pneumoconiosis among workers in cement and plaster but did not include proportional statistics. He believed that inhalation of cement dust did not produce tuberculosis and did not favor the progress of the disease. Rota and Finzi found that of 63 deaths among the permanent workers engaged entirely in lime and cement work 28 died before the age of 40 and 11 under 50. The cause of death in 20 of the 63 cases was pulmonary diseases - 9 acute pneumonia, 2 pleuromediastinitis, and 9 tuberculosis. Rota and Finzi stated that there was no truth in the statement that workers in limekilns enjoyed a certain immunity from tuberculosis. On the contrary, they found that the greatest toll to this disease was paid by kiln workers. For example, 9 of 35 deaths of such workers were due to tuberculosis. Physical examination of 218 factory workers revealed 132 cases of harsh respiration in the upper respiratory passages, due certainly, they asserted, to an incipient pneumoconiosis or infiltration of dust into the peribronchial lymphatic channels. Harsh respiration was found more frequently in those employed for a long time, especially those who had worked 10 to 12 years; it was present in 52 of 128 kiln workers, 18 of 26 workers transporting stone, 43 of 50 crushing-machine operators, baggers, and porters of sacks. Many workers were also emphysematous.

Bianchi (78) made a careful clinical and radiological examination of 250 workers employed in more or less closed workrooms, as sculptors, rough hewers, modelers, and workers in grinding rooms. After excluding all cases of suspected syphilis and other chronic lesions, they studied 73 workers in marble who had no inherited disease or previous disease history and were free from definite or suspected symptoms of affections of the respiratory passages which might come under the category of common infections. X-ray examination revealed diffuse nodes of thickened tissue spread over almost the entire respiratory surface. In these cases he could determine only a slight percentage (20 percent) of functional alterations with symptoms of chronic bronchitis and pulmonary emphysema.

As the result of a radiographic and clinical examination of 105 Carrara marble workers employed in marble grinding and of other workers such as sculptors, rough hewers, sawyers, and polishers Turano (79) made the following statement:

> The changes met with among Carrara marble workers may be classed in the initial stage of pneumoconiosis, that is to say, comprising the least serious forms of the disease, such as the very marked reinforcement of the pulmonary outline, due as demonstrated by personal observations, to the processes of arteritis and lymphangitis, and in certain cases equally to conditions of emphysema usually present among those workers.
>
> I have also found frequent pleuritic changes which can be related to inhalation of marble dust, but never lesions of the pulmonary parenchyma.
>
> The radiological aspect of pulmonary tuberculosis among workers examined is on the other hand highly important, since it shows an atypical picture on which are noted lesions with unusual sites and apical and subclavicular regions unaffected. It is this fact which has led me to admit the probability of a combination of pneumoconiosis and tuberculosis.
>
> Finally, statistical as well as radiological data justify absolute exclusion of the theory of a particular benign or malignant course of tuberculosis among marble workers, as likewise of any kind of predisposition to the said specific disease.

South Africa

Mining started in South Africa on the Witwatersrand in 1886 (80). At first the workings were shallow and in the oxidized or "free-milling" zone where the rock was relatively soft, friable, and damp. By 1892 extensive deep-level properties had been operated, and drilling by machines was introduced. As long as the mines were working in the free-milling zone very little dust was produced, as the mines were shallow and probably fairly well-ventilated, and it is unlikely that the men were greatly affected by the dust. Very few people in South Africa suspected that mine dust was in any way injurious to health until the Government mining engineer of the Transvaal Mines Department in a report in 1902 mentioned "miners' phthisis" as a disease "which seems to be peculiar to men employed in rock-drill work." This report, which covered the 6 months ended December 1901, stated that of 1,377 machinemen employed previous to 1899, 225 were known to have died between

October 1899 and January 1902, an average annual death rate of 73 per 1,000. These facts arrested the attention of the Government, the mining community, and the general public, and in December 1902 the first (Transvaal) Miners Phthisis Commission was appointed "to inquire into and report on the disease commonly known as miners' phthisis." A report was issued in 1903. The commissioners attempted to procure a medical examination of all working miners, but the returns were incomplete partly because many of the miners were reluctant to submit themselves to examination. Of the 1,201 miners examined 15.4 percent were affected by miners' phthisis and 7.3 percent were suspected. The Report of the Medical Commission issued in February 1912 was the second landmark in the medical history of silicosis in South Africa. The actual examinations were more complete than in 1903 but did not cover all the underground employees. A general clinical examination was made of 3,136 working miners, supplemented by a special examination of 326 men, in which radiography was for the first time applied to the examination of cases on a fairly extensive scale. The prevalence of the disease among the working miners examined was found to be 26 percent, with an additional 5.5 percent of doubtful cases. This figure is somewhat higher than that found in 1903 but probably reflects the result of a more extensive investigation.

According to Irvine, Mavrogordato, and Pirow (81) 1916 was a cardinal year in the history of silicosis on the Rand. It was marked by the institution of the Miners' Phthisis Medical Bureau and the publication of the General Report of the Miners' Phthisis Prevention Committee. The present-day system of detection and prevention of silicosis on the Rand dates from 1916. A total of 6,472 original compensation awards were made to miners for silicosis from 1912 to 1916.

According to the Report of the Miners' Phthisis Medical Bureau (82) for the Three Years Ended July 31, 1932, the average number of new cases of silicosis and tuberculosis with silicosis among the miners of the Witwatersrand was over 800 annually during the 4-year period 1912-16. The report also states that 895 cases of "primary-stage" silicosis were detected from 1917 to 1920; 728 cases of "anteprimary stage" from 1920 to 1923; 1,235 cases of "anteprimary stage" from 1923 to 1926; and 917 cases of "anteprimary stage" from 1926 to 1929. In 1930-31 the number of new cases for the first time in 8 years fell below the standard level of 251 cases, which was the average annual production for the period 1920-23.

The following statement by Hildick-Smith (83) summarizes briefly the status of miners' phthisis in South Africa. After quoting figures showing the percentage production rate of silicosis for the preceding 15 years he said:

> From a study of these figures I personally am inclined to think that the time is not far distant when the incidence of silicosis will have ceased to be a matter of serious concern to the industry. This, I think, will be accomplished by the elimination, in whole or in part, of the dangerous dust from the underground air. Old habits of thought were inclined to persist in regard to the production of silicosis on the mines. The report on the work of the Miners' Phthisis Medical Bureau showed the

> maximum percentage production rate for silicosis had decreased from 14.5 percent after 14 years' service in the period 1918-20 to 4.5 percent after 17 years' service in 1931-32. The graduation rates for all miners, including men working underground before the inception of the Phthisis Bureau in 1916, show that the present maximum production rate is under 4 percent after 20 years' service. Of the 157 miners notified last year that they had contracted silicosis more than one half began work in 1916-17, one in 1922-23, and one in the following year. Since 1923 there have been no cases of contracted silicosis - in other words no new Rand miner has been found suffering from silicosis in the last 10 years. These results are confirmed by the fact that less than 50 percent of the patients at the Springkell Sanatorium are of the miner class.

With regard to the employment of silicotics, Hildick-Smith said that 978 are now employed by the mines - an increase of about 110 since November 15, 1933.

United States

Data are not available on the general incidence of silicosis in the United States. Such data are difficult to obtain in the existing state of American vital statistics; exact knowledge of the population at risk in a given occupation, classified by age, is obtainable only with great difficulty and by special and intensive research (37). In an analysis of statistical data of this kind Greenburg (37) calls attention to the importance of considering groups that are fairly comparable, so that the effect of industrial hazards will not be complicated by the influence of social and economic factors of a more general nature. He states, for example, that in Hoffman's studies published in 1918 ratios presented for the various dusty trades are based on the industrial experience of the Prudential Insurance Co. but that the ratio used as a norm for comparison of males in the registration area was obtained from data of the United States Census Bureau. On this basis almost all the industries which he tabulates show a surprisingly high tuberculosis ratio, including many trade designations of workers, such as "iron and steel workers", who can hardly be considered as generally exposed to a serious dust hazard; his abnormally high ratios, therefore, probably are due to the general social and economic conditions of the wageearner's life and to the fact that the group is an industrial one. It seemed evident to Greenburg (37) that a comparison between the Prudential figures for a given dusty trade and the Census figures for all occupied males, which gave Prudential ratios 25 to 50 percent higher for tuberculosis than those for the registration area, was not a fair one and that the conclusion reached through such a comparison was unwarranted.

Several special investigations, carried out in certain industries in the United States, that have attracted attention because of the high death rate from tuberculosis (84) are described below.

Mining.- The first investigation of silicosis in the mining industry in the United States was made in 1914-15 by the United States Bureau of Mines in cooperation with the United States Public Health Service in the Joplin

(Mo.) mining district (85). Of 93 men examined 64 showed plain and definite evidence of pulmonary disease, 3 were suffering from nonpulmonary disease, and 26 were seemingly well. To gain a more accurate idea of the prevalence of consumption among the miners in the Joplin district it was decided to examine a large number, and from May 15 to December 31, 1915, 720 miners were examined; 45.7 percent had silicosis and tuberculosis and 5.3 percent had tuberculosis. A later report (1927) of this same investigation by Lanza and Childs (86) contained the first detailed X-ray studies of silicosis made in the United States.

Investigations of mining conditions by Harrington and Lanza (87) in Butte, Mont., in 1921, revealed that 42.4 percent of 1,018 miners examined showed definite signs of lung damage due to dust. An examination in 1921 of 303 gold miners in Nevada disclosed that 80 percent had silicosis, and a study in California in the same year revealed that 25 percent of 181 gold miners had silicosis.

In 1923 the mining companies of the Tri-State district (comprising the zinc- and lead-mining areas of southwestern Missouri, southeastern Kansas, and northeastern Oklahoma) asked the United States Bureau of Mines to determine whether measures in use were adequate for the prevention of silicosis and, if not, to recommend improvements. The 1923 investigation (88) included the examination of 309 miners, 101 of whom were found to be negative, 114 doubtful, and 94 positive cases of silicosis. Of the positive group 52 were in the first stage, 22 in the second stage, and 20 in the third stage of silicosis.

From July 1, 1927 to June 30, 1932 the Metropolitan Life Insurance Co., the Tri-State Zinc & Lead Ore Producers Association, and the United States Bureau of Mines operated a cooperative clinic at Picher, Okla. (89), to demonstrate to industry a workable method for diagnosis of silicosis, to educate workers in preventive measures, and to serve as a model to industries presenting a dust hazard. During this period, of 27,553 individuals examined, 5,366 had silicosis, 742 silicosis plus tuberculosis, and 320 uncomplicated tuberculosis.

According to the United States Public Health Service (90) the percentage of anthracite miners and their helpers showing signs of pneumoconiosis increased from 2 percent among those with less than 5 years' service to 16 percent among those with more than 15 years' service (for persons under 45 years of age). Of 95 X-rays of bituminous-coal miners 40 (42 percent) showed generalized fibrosis, chiefly linear in character. The percentage of deaths caused by all respiratory diseases among anthracite miners in Pennsylvania was definitely higher than that of other adult males in the general population - 57.6 percent in contrast with 37.2 percent among other men of the same age in the Wilkes-Barre coal field (91).

Kibbey (92) reported in 1931 that practically 100 percent of the miners who worked in the several coal mines operated by his company in Alabama had anthracosis and that they had a higher mortality rate from tuberculosis than any other wageearning group.

From a study of industrial morbidity statistics for the period 1924-27 Bloomfield (93) found that 38 cases of pneumonia had occurred among the 1,637 bituminous-coal miners employed during the same period in the mines operated in connection with an iron and steel plant. Occupational analysis disclosed that 33 of the 38 pneumonia cases were associated with only 2 of the 69 different occupations in the mines - pick-mining and loading coal. The pneumonia rate per 1,000 for miners and loaders was 31, whereas the rate for all other mine workers was only 8.5 per 1,000.

The apparent similarity of the risk in mining and the risk in the processes involved in excavating and tunneling, so far as the silicosis hazard is concerned, led to an investigation of such processes in New York City by Smith and Fehnel in 1929 (84). Of the 208 drillers, blasters, and excavators examined 42 percent showed early and 15 percent well-developed silicosis. Evidence of tuberculosis, including both active and inactive cases, was found in 9 percent of the total number.

Grinding.- According to Hoffman (4) the industrial insurance mortality statistics of the Prudential Insurance Co. from 1897-1914 showed that 143 (46.9 percent) of 305 deaths among grinders were due to pulmonary tuberculosis. In another group of 5,988 grinders, which included cutlers, scissors grinders, ax, plow, and other steel grinders but excluded foremen and superintendents, the actual mortality from all causes was 17 percent in excess of the expected mortality; in other words, for every 100 deaths expected on the basis of normal experience there were 117 deaths among this group.

In 1920 Winslow and Greenberg (94) investigated the dust hazard in an ax factory. They quoted Drury's exhaustive statistical study on the incidence of tuberculosis among grinders and polishers as showing that from 1900-18 the death rate in a group including 90 polishers, 85 wet grinders, and about 25 dry grinders was 1,000 percent. Their investigation revealed dust conditions serious enough to cause such an excessive death rate.

To indicate the effect of silica as a predisposing cause of tuberculosis Riddell (95) in a paper published in 1926 quoted the following mortality table prepared by Drury for a Connecticut community (agricultural except for an ax factory employing 800 men):

Death rate from tuberculosis in a Connecticut community

Entire population of factory district	200 per 100,000
State as a whole	150 per 100,000
Factory population	650 per 100,000
Polishers and grinders	1,900 per 100,000

In a review of the pulmonary tuberculosis developed in a large grinding-wheel plant Clark (96) found that from January 1, 1918 to December 31, 1930 there had been 42 cases of active pulmonary tuberculosis, 38 of whom were men and 4 women; 37 of the group were employed at factory work of some kind, while 5 did clerical work. Of the 42 workers who had developed tuberculosis

20 were dead at the time Clark prepared his report, 18 were living, and 4 could not be traced. Of the 18 living 13 were working, 3 were at home under care of a physician, and 2 were in hospitals. The average number of employees during the 13-year period was 2,460.

According to Kessler (97) over 100 cases of silicosis were alleged to have occurred in the abrasive-powder industry in one State involving 4 companies which pumped sand by hydraulic pressure to a plant, where it was washed, steam-dried, screened into various sizes, and then pulverized in closed tube mills. Six of the workers had died and been autopsied and many others were reported to have died, but the results of the autopsies were not known.

Granite industry.- In his report of dust phthisis in the granite-stone industry, published in 1922, Hoffman (98) summarized the results of his investigation as follows:

> The granite-stone industry is carried on by wageearners who, broadly speaking, live under sanitary conditions above the average, so that possibly unfavorable environmental factors are of decidedly secondary importance.
>
> The housing conditions under which granite workers live are also above the average, so that in this respect the environmental factors are favorable to a low mortality rather than otherwise.
>
> Anthropometric records clearly establish the fact of a superior physique, indicative of a higher degree of disease resistance, as determined by a relative weight above the average. From this point of view, therefore, granite workers should experience a relatively low mortality from pulmonary tuberculosis instead of a mortality decidedly above the average normal to industrial occupations.
>
> Granite workers, considered by specific occupations show wide variations in tuberculosis frequency, the excess in the death rate being most marked among the men employed in granite-stone cutting, it being especially severe among men employed in the use of pneumatic tools. Certain occupations, such as polishing, tool sharpening, bed setting, etc., do not show a marked excess, if any, in the mortality from pulmonary tuberculosis, clearly indicating that the risk is practically proportionate to dust exposure.
>
> Compared with the normal death rate of adult males of the State of Vermont, or of New England, the mortality from pulmonary tuberculosis among granite-stone workers has increased enormously during the last 2 years, as contrasted with a diminishing mortality in the population at large. Against a decrease in the pulmonary tuberculosis death rate of adult males of the State of Massachusetts from 288.5 per 100,000 exposed to risk during 1895-99 to 203.2 during 1915-18 there had been an increase in the corresponding death rate of granite cutters of the

New England States from 432.0 per 100,000 during 1895-99 to 1056.7 during 1915-18. The only other occupation for which information is available for the corresponding period of time is that of glass-bottle blowers, among whom the mortality from pulmonary tuberculosis diminished from 418.6 per 100,000 to 265.9. These statistics for the New England States are confirmed by similar data for every other stonecutting center of the United States, proving with absolute certainty that in every section of the country the tuberculosis mortality of this group of industrial workers is increasing, in contrast to a locally diminishing death rate from this most fatal of all diseases * * *.

Recalling that the normal pulmonary tuberculosis mortality of adult males in Massachusetts is only 203.2 per 100,000, it is shown that the present death rate from pulmonary tuberculosis among granite cutters is 5 times the normal experience in the population at large and probably 6 times what it should be on the basis of strictly noninjurious occupations carried on largely under hygienic conditions and in the open air.

The same conclusion applies to nontuberculosis respiratory diseases, for it is shown that the mortality from bronchitis, pneumonia, and asthma is also on the increase among granite cutters, in contrast to a diminishing rate of frequency among adult males of the general population.

In the second series of investigations on health of workers in the dusty trades the United States Public Health Service studied the granite industry at Barre, Vt. (99). The occupational groups were divided into four general classes based on the amount of dust to which they were exposed. These groups consisted of:

(A) Hand pneumatic-tool cutters, 614 persons, exposed to an average of approximately 60 million particles.

(B) All other occupational groups exposed to more than average plant dustiness. This group contained 104 persons in occupations where the dustiness averaged between 27 and 44 million particles.

(C) Those occupational groups consisting of 146 persons exposed to average plant dustiness (20 million particles).

(D) Those occupational groups exposed to less than average plant dustiness. This group contained 108 persons in occupations where the dustiness averaged between 3 and 9 million particles.

The following summarizes in part the results of the study on prevalence of silicosis and tuberculosis among these workers:

Having a clear-cut diagnosis of silicosis and complete information with regard to the magnitude of dust exposure, it was possible to study the proportion of persons developing silicosis by length of service. In the A group, or that group in which there was heaviest dust exposure, the first case of silicosis appeared after approximately 2 years of service, and by 4 years of service all in this group seemed to have developed at least an early case of silicosis. In the same dust-count group the first case of more developed silicosis appeared after 5 years of service, and by 9 years approximately 90 percent had advanced to this stage. The study of the other dust-count groups showed that the development of silicosis was proportionate to the dust exposure. In the case of the D group, 2 cases of early silicosis occurred after 10 years' exposure, and 1 case of moderately developed silicosis after 6 years' exposure.

General prevalence of tuberculosis complicating silicosis.- In plants carrying morbidity records, where it was believed certain that all cases of tuberculosis were diagnosed as such, the total rate was 5.7 percent or 6.5 percent if early cases are included. Latent or suspected cases of tuberculosis (among those given physical examinations) gave an additional 3.8 percent. These figures represent the number of cases found over a period of $2\frac{1}{4}$ years, and consequently the rate is different from what would be obtained on an ordinary cross-section survey.

Tuberculosis by dust groups and years in granite.- The association between the high tuberculosis prevalence among granite workers and the dust hazard was shown by determining the rates for that disease in the different dust-count groups by length of service in the granite industry. The comparison between A and B groups and C and D groups was extremely significant. No cases were found in the D group, and C group furnished but 3 cases, 1 of which occurred under 5 years' exposure; another, under 10 years. Of particular importance is the fact that in 35 years of exposure the rate did not rise. Rates for the A and B groups rise steadily with increase of length of service to 15.5 percent in group A and 19.1 percent in group B. Even when the suspected tuberculosis cases are added, the general picture is not materially changed. In neither the C or D groups does there occur any excess of cases after long exposure, while in the A and B groups the rate rises with length of service in a similar manner as for active tuberculosis.

According to Goodrich (100) statistics show that workers exposed to silica dust and, to a smaller extent, other dusts have an excessive death rate from tuberculosis. The introduction of pneumatic and electric tools in processing stone has caused a tremendous increase of tuberculosis of the lungs in those working with these appliances. In 1890 when stone was cut and polished with comparatively crude tools the death rate from tuberculosis among these workers was 150 per 100,000. By 1910 it had increased to 1,080 per 100,000 and by 1925 had reached 1,950 - an increase to 13 times that of the period before high-speed tools came into use. The explanation, of course, is that modern equipment creates many times the density of dust in air produced by manual labor. Cases of silicosis also became very numerous, and many of them developed tuberculosis. During this same period, when tuberculosis was increasing so rapidly among these workers, the death rate from tuberculosis throughout the country was reduced to about half the rate of 1890.

The report of the Special Industrial Disease Commission of Massachusetts (101) shows that the granite industry presents a severe silicosis hazard. Surveys were made of 314 granite establishments, and dust studies were made in 13; X-rays and physical examinations were made of 961 graniteworkers; 15.2 percent were found to have silicosis and 7.6 percent silicosis with tuberculosis.

Foundry industry.- The Special Industrial Disease Commission of Massachusetts surveyed 225 foundries (101), in which 12 dust studies and 1,614 physical examinations and X-rays were made. In the foundries studied excessive dust counts were found in connection with many of the operations. The need for immediate, effective control of dust conditions in foundries was evidenced by the fact that silicosis was found in 8.8 percent and silicosis with tubersulosis in 2.6 percent of the workers examined.

In a study of the foundry industry McConnell and Fehnel (102) found that the death rate for respiratory diseases (including all forms of tuberculosis and influenza) for iron and steel foundry workers was about two and one third times that for workers in all industries combined and was more than twice that for workers in any of the industries selected for comparison. More than a third of all deaths among foundry workers were caused by some respiratory disease, whereas but little more than a fifth of the deaths of workers in industry were due to these causes. A striking fact revealed is the uniformly high death rates for foundry workers for each of the respiratory diseases throughout the working period of life. Another outstanding fact is the high rate for pneumonia, bronchitis, etc., the foundrymen's rate being nearly three times that for all workers combined. Among iron and steel molders, founders, and casters, where 12 deaths from pneumonia might have been expected, there actually were 38, a ratio of actual to expected deaths of 315 percent; 24 deaths were recorded for respiratory tuberculosis, where 13 might have been expected, a ratio of 179 percent; the ratio for influenza was 216 percent, based on 23 actual compared with 11 expected deaths. In an analysis of the occupational mortality of adult white male industrial policyholders of the Metropolitan Life Insurance Co. who died during 1922-24 a comparison is made of the proportion of deaths due to any one cause in any one specified occupation group with the proportion due to the same cause among all occupied white males. Again, pneumonia was the leading cause of death among foundry workers, a condition not true of any of the other 71 occupations included in the analysis. When differences in the age composition of the two groups were taken into account it was determined that the proportion of deaths from pneumonia was 120 percent higher for foundrymen than for occupied males generally. Likewise, the proportion of deaths from influenza was 82 percent higher, that for tuberculosis of the respiratory system 5 percent higher, and that for other respiratory diseases 17 percent higher. McConnell and Fehnel (102) emphasize the harmful effects on the lungs of exposure of workers to dust inhalation; 67 of 215 X-rays taken were diagnosed as positive for silicosis. Advanced cases of silicosis were not found among those examined, but sufficient evidence of occurrence of the disease was presented.

Cement industry.- A 3-year study of the cement industry by the United States Public Health Service (103) revealed the following:

> X-ray films were obtained of the chests of 53 employees in several different occupational groups, length of service varying from 6 months to 15 years. On the basis of the findings, these employees were classified into 5 groups, as follows: (1) Those showing evidence of pneumonoconiosis, (2) those showing evidence of tuberculosis and pneumonoconiosis, (3) those negative for pneumonoconiosis, (4) those showing evidence of tuberculosis without pneumonoconiosis, and (5) doubtful cases.
>
> The earliest case of pneumonoconiosis appeared after 3 years' exposure to cement. Of the 53 men selected, 37 had been in the industry more than 3 years and are therefore used as a basis of comparison. Among these 37 men, 15 showed evidence of pneumonoconiosis, 3 of the 15 also having tuberculosis. In the group of 22 who showed no evidence of pneumonoconiosis 8 gave evidence of arrested tuberculosis, 4 showed no evidence of either pneumonoconiosis or tuberculosis, while 10 were considered doubtful.
>
> In none of the cases showing pneumonoconiosis were there any clinical symptoms of the condition, although the general fibrosis present and its distribution through the lungs were indicative of the condition. Cement workers appeared to have more than a normal amount of calcified nodes in the lungs.
>
> Of the 570 workers examined, 21, or 3.7 percent, were diagnosed as either positive or suspected cases of pulmonary tuberculosis. In only 2 cases, however, was the disease active at the time of the first series of special chest examinations, and neither appeared to progress as the result of exposure to the dusts. In the history of both cases it was evident that the disease had developed before the men entered the cement industry. One of them died about a year after the study closed, while with the other the disease appeared to have become quiescent when the study was closed. All the rest of the cases, with the exception of three, which were doubtful as to diagnosis, appeared to have developed their lesions before they entered the industry and were continuing in their occupations without any evidence that the lesions were progressing.

According to Russel (104) the frequency of disability on account of respiratory diseases among the cement workers was twice as great as the average respiratory rate among employees of 11 manufacturing plants in relatively nondusty industries. The highest rate for all respiratory diseases in any one of these establishments was 30 percent below the rate for cement workers.

Canada

Elliott (105) was the first to direct attention to silicosis in Ontario, Canada; in an article on Silicosis in Ontario Gold Mines published in 1924 he reported that 3 of 11 underground workers examined by him presented evidence of silicosis, indicating that the disease was being produced in this mining area. In 1925 and 1926 a further survey was made by the Industrial Hygiene Division of the Ontario Department of Health. Miners with more than 5 years' experience underground in the individual mining area and with no exposure to silica dust elsewhere (a few of those examined had had some exposure elsewhere) were given a physical examination. Of 1,220 men examined 74 showed evidence of silicosis - 52 in the anteprimary, 11 in the primary, and 11 in the secondary stage.

During the period April 1, 1926 to April 1, 1927 a survey of silicosis was made in the Porcupine group of mines in Northern Ontario under the auspices of the mine operators in compliance with the amendment to the Miners' Phthisis Act of April 8, 1926, bringing silicosis under the status of a compensable industrial disease. Approximately 4,000 miners were examined for tuberculosis, silicosis, or both. As a result, 94 silicosis claims were submitted for compensation. Using the South African nomenclature, of these 94 cases 39 were diagnosed as anteprimaries, 28 as primaries, and 27 as secondaries; in addition, 247 men showed either positive or probable tuberculosis (106).

Types of Dust Injurious to Health

The relation between dust inhalation and lung diseases was recognized very early, as indicated by the literature cited above, but it was also recognized that certain dusts caused severer symptoms with quicker fatal issue than others. As the symptoms caused by inhalation of certain dusts were long delayed or were attributed to other causes, such dusts were considered harmless and some even beneficial in the prevention of lung diseases when inhaled with the more harmful ones. Ancient writers seldom distinguished between the various forms of respiratory diseases but rather referred to a general relation between lung affections and dust inhalation (28); and no really scientific investigations were made of the workers or the dust. Ramazzini, however, described the effects on workers of various types of dust produced in such industries as the hemp and flax industry, silk-combing, and corn-sifting, in addition to mining and similar industries. According to Collis (28) the distinctions, implied or definitely stated, in Ramazzini's excellent clinical description of the types of respiratory trouble which follow inhalation of different dusts

> are the more notable, because even today pneumonoconioses are pigeonholed in clinical teaching as a single entity, ascribed to exposure to any and every form of injurious dust, of which pulmonary fibrosis sums up the pathological findings and phthisis the morbid result.

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In the decade preceding 1863, when the study of public health was organized in England (28), Greenhow made an elaborate statistical inquiry into the influence of occupation on health in connection with lectures on public health at St. Thomas' Hospital. Simon, then medical officer to the General Board of Health, considered Greenhow's investigation so important that his report was published by the board, and shortly after when Simon became medical officer to the Privy Council he intrusted Greenhow with the duty of further investigations in the great industrial centers. The resulting reports are, according to Collis (28), the first example of State medical inspection of factories, and he makes the following statement regarding them:

> Throughout these reports runs as a theme the influence of dust inhalation in causing pulmonary disease, whether among lead miners in Yorkshire, tin miners in Cornwall, needle pointers in Alcester, cotton operatives in Lancashire, flax hecklers in Pately Bridge, metal grinders in Birmingham and in Sheffield, coal miners in South Staffordshire and in South Wales, or stone dressers in Stroud. Why work so well started was then allowed to lie dormant for so long, while other aspects of public health were being strenuously developed by medical officers of health with inspectors of nuisances appointed for every town and district, reinforced now by a battalion of tuberculosis officers, is astonishing.

As a result of investigations in South African mines and in other mining districts of the world, so much attention was focused on one particular dust - silica - as the most harmful encountered in industry that most investigators had accepted other dusts as harmless or of negligible importance as a health hazard in industry and the disease - silicosis - resulting from breathing silica dust as the only important dust disease. Other dusts, such as the silicates (asbestos, for example), have been found almost as harmful as silica dust, but the effect on the lungs is somewhat different from that of silica and the hazard not so widespread.

From general underground experience for more than 25 years in coal and metal mines and from an intensive study of dust for 8 years Harrington (107) concluded that -

> Any dust insoluble in the fluids of the respiratory passages and in sufficiently finely divided form to float in the air and be breathed by underground workers will ultimately be harmful to health if the dust is in the air in large quantities and is breathed by workers for considerable periods of time. This applies to insoluble nonmineral as well as mineral dusts or mixtures of them and includes coal dust or mixtures of coal and other dusts. There are also some definitely harmful mine dusts which are soluble, and some dust experts appear to believe that the so-called insoluble dusts under certain conditions become soluble and are harmful only when soluble.

An outstanding impression gained by Ballantyne (108) from the reports of the International Conference on Silicosis was that silicosis constitutes only one facet of a very large question - the prejudicial effect of dusts in general upon the human subject. He concluded that -

> It (silicosis) is only one of several close-related pulmonary affections, due to dusts of different kinds. It is the most important of these, but only because it is the most definitely known and so many workpeople are exposed to the risk. In the case of asbestos dust knowledge regarding the injurious effects of inhaling it has advanced so far as to justify the adoption of legislative measures analogous to those relating to silicosis. There are many other kinds of dust, however, as to the effects of which, when inhaled, we have little or no knowledge, and the investigation of which calls for early attention.

The Report of the Silicosis (Medical Arrangements) Committee issued in London in 1929 (109) contains the following statement:

> We are convinced that silicosis is more widespread than is generally believed and that it occurs to some extent in a number of industries and occupations where its presence has not been suspected.

In 1930 Kettle (110) called attention to the harmfulness of breathing dust:

> Dusts of various kinds occur in the atmosphere in all parts of the world, and the inhalation of these dusts over periods of years inevitably produces changes in the lungs. Everyone is familiar with the pigmented lung of the city dweller, an example of a pathological change produced by the inhalation of dust which is of no clinical importance whatever, but an exaggeration of this condition in the coal miner causes definite symptoms, and when the inhaled dust is not the relatively harmless carbon, but one of the much more sinister possibilities, serious pulmonary changes develop. The pneumonoconioses have received less attention than they deserve, even in textbooks of medicine and pathology. But to the doctors whose patients are exposed to dusty trades, to those responsible for the conduct of these trades, and to the factory inspector, pneumonoconiosis is of great moment.

In the report of a study of dust conditions in German industries, published in 1932, Lochtkemper and Teleky (66) stated in the summary:

> The breathing of any dust leads to injury of the lungs, if it is intensive enough and continued long enough.
>
> With intensive and long-continued inhalation of dust, entirely quartz-free dust leads to lung changes clearly recognizable in the X-ray picture. The blood vessels appear more clearly defined; there are also more numerous and thicker honeycomb formations, also infiltrated mottling which, however, never shows the very sharp delineation and density caused by quartz. The subjective symptoms referable to the changes are slight; there is none of the lung rigidity characteristic of silicosis but a complication of diseases, especially bronchiectasis and emphysema.

It is theoretically possible from the report that many of the occupational dusts breathed in immense amounts over a very long time lead to changes not recognized by us today.

In a discussion of the etiology, pathology, and physical signs of silicosis Levey (111) stated:

> However, a lung filled with dust is a wounded lung and should be considered as subnormal in its vital resistance toward the onset of disease. A lung containing silica dust has a strong predilection toward the onset of tuberculosis. This susceptibility is unusual.

Terminology of Dust Diseases

The following terminology of dust diseases of the lungs (112) is now used by most writers on the subject:

Pneumoconiosis.- A general term covering all dust diseases of the lungs, fibrous or nonfibrous (from Greek pneumon, lung, and konis, dust).

Silicosis.- A fibrosis caused by free silica (or quartz) and the best known scientifically of the dust diseases of the lungs.

Silicatosis.- A type of fibrosis found after exposure to certain mineral dusts and assumed to be caused by various silicates. It is distinct from the sharply defined, coarse, nodular fibrosis caused by silica dust.

Anthracosis.- A dust disease of the lungs found in coal miners; it is ill-defined and is presumed to depend on inorganic dust in coal. The lungs are black.

Siderosis.- A term applied to a fibrosis of the lungs found in metal workers. The condition is ill-defined. The lungs are yellow or red from metallic oxides, generally of iron.

Asbestosis.- A fibrosis of the lungs with characteristic microscopical stigmata due to breathing asbestos dust, a silicate of magnesium.

Pneumoconiosis

The diseases resulting from the inhalation of dusts generated in industry during the progress of certain processes are generically known as pneumoconiosis, having been so named by Zenker, as mentioned previously. In a brief summary of information regarding certain of the pneumoconioses other than silicosis Collis (113) called attention in 1931 to some of the respiratory diseases that may be caused by dust but seldom are recognized as dust diseases. The inorganic dusts he classed as insoluble, soluble and harmful, and mixed. Under insoluble dusts he mentions that those from certain materials, such as basic slag and emery, an oxide of aluminum, are insoluble in the tissues. Inhaled particles fall on the walls of the bronchi and bronchioles, where they are entangled in secreted mucus and then swept back by ciliary action to be expelled finally in sputum. If the dust exposure is excessive over a period of time an inflammatory hyperemia of the walls results, with an excessive exudation of mucus; finally the process extends beyond physiological elasticity; then degeneration and destruction of the ciliated mucosa occur; microbic invasion follows; and chronic bronchitis is established.

The process described by Collis is a reaction to the inhalation of all dusts not rapidly absorbed; hence bronchitis stands out as chief of the dust diseases and during middle life causes much recurrent invalidity and incapacity and, after middle life, high death rates. Dust bronchitis cannot be distinguished clinically from bronchitis due to exposure at hot furnaces, fumes in industry, or severe climatic conditions; hence it association with dust inhalation lacks recognition.

If the dust particles are small enough (5 microns or less) to be drawn into the finer bronchioles and alveoli, which are the seat of attack for pneumococci, a similar reaction is stimulated; hence the resistance of these parts to infection is lowered, and pneumonia results. Pneumonia is even less-recognized as a dust disease than bronchitis; nevertheless, mortality records of those employed in dusty occupations are high. However, when fine particles of insoluble dusts are carried by phagocyted dust cells from the alveoli into the lymph stream and lymph nodes of the lungs they tend to remain there as foreign bodies and do not provoke any particular tissue reaction.

Collis (113) says that the most-studied dust of the soluble and harmful dusts in this group is that of silica. Next come the dusts of silicates; although many silicates, such as fire clay and pottery clays, appear to exert little if any harmful influence upon the lungs recent work has shown that certain other silicates, such as basalt and asbestos, react injuriously on the pulmonary tissues. The reason for these differences is not clear; but probably it lies in the constitution of the various silicates, the SiO_2 radical being less firmly attached to the molecule in some cases than in others.

Collis (113) defines asbestosis as a pneumoconiosis that advances to a fatal end without the supervention of any characteristic infection. It is a simple dust condition, just as is simple silicosis; but it is more distressing in life and more rapid in this progress than silicosis. It contrasts even more strongly with pulmonary mycosis, which is due to a living infection upon otherwise healthy lungs.

Regarding the mixed dusts, Collis (113) says that as long as any dust consists of only one substance its influence can be isolated and studied; but the case is different when more than one substance is present in the dust. Coal dust, which recently has been carefully investigated (114), is taken as an instance in point. No undue mortality from respiratory diseases is said to be found among coal miners as long as the coal worked contains little or no mineral matter - presuming that the miners are not exposed to other dusts, such as those arising from rocks intervening in the coal measures. The miners in the Nottinghamshire coal fields furnish a good example; the lungs of these men may be as black as the coal they work, but they remain resilient and free from fibrosis.

In discussing the nomenclature of dust diseases Pancoast and Pendergrass (22) state that although silica (SiO_2) seems to be the active fibrosing agent in most conditions recognized as essentially a pulmonary fibrosis adherence to the general term "pneumoconiosis" still seems advisable. Some other dusts undoubtedly cause a fibrosis directly, and still others may either tend to retard the action of silica, as coal dust and clay, or enhance its action, as alkalies in the mixtures of silica and powdered soap in scouring powders. Silicosis is the word much more frequently used, mainly because it is not so unwieldy and usually describes accurately the particular phase of the condition under discussion. "Miners' phthisis" appears in the laws of South Africa but is otherwise an obsolete term generally recognized as implying silicosis or pneumoconiosis. There is now reason to believe that silicates also produce considerable fibrosis through their silica content or because certain silicates have a specific action of their own.

According to Pancoast and Pendergrass (22) there is even more reason to employ the occupational terms now than in the past because recently certain peculiarities have been attributed to the dusts of those industries, even though the condition still remains silicosis or pneumoconiosis. For example, in anthracosis the action of the silica seems to be modified by the coal dust mixed with it; moreover, coal dust alone may produce a mild fibrosis. Collis and Gilchrist (114), in a recent investigation of coal trimmers working at the Cardiff docks, found that inhalation of large amounts of coal dust alone over a period of years produced evidences of mild pneumoconiosis which presented the same roentgenographic appearances as silica. It has been suggested that clay has a similar effect in protecting the workers in some of the operations in pottery industry, but not all of them. The term "chalicosis" has been applied in the past to pneumoconiosis of potters.

In recent years certain very unusual features in connection with pneumoconiosis among asbestos workers have brought the term "pulmonary asbestosis" into active use.

According to Haldane (115) evidence is accumulating constantly that scattered fibrosis, giving essentially the same X-ray picture as early silicosis, is comparatively common as a result of excessive dust inhalation, but without accompanying liability to tuberculous infection. However, he thinks that this condition should not be called silicosis, and since it seems to arise from excess of any kind of dust and not merely silicate dust pneumoconiosis seems the most suitable name for it.

Silicosis and asbestosis are forms of pneumoconiosis that have been clearly defined clinically; the other forms of pneumoconiosis, as anthracosis, siderosis, and many others, still lack complete definition. Although considerable radiographic evidence has been accumulated on these latter types of occupational dust exposures corroboratory evidence has not been deduced from correlations of the X-ray findings with pathological anatomy, chemical examination of tissues, and studies of the industrial environment (116).

Silicosis

Definition

Rovida (117), the first to mention silicosis, used the term in 1871 in describing "a case of silicosis of the lungs with chemical analysis."

At one time it was believed that several substances were capable of producing the lung condition called silicosis, but it is now agreed fairly generally that all types of dust fibrosis of the lungs are due to some form of the element silicon (118). The recognition that silicon was the important element in pneumoconiosis was a great step forward but, according to a recent statement by Kettle (118), there is no doubt that the teaching that only one form of silicon - the oxide silica - is capable of producing the disease was accepted too readily. The realization in recent years that asbestos can produce serious pulmonary fibrosis should have been a warning that a too-restricted view was being taken of the matter; this has been borne out by the recent work of Jones on sericite. However, the following definition adopted at the International Silicosis Conference in South Africa in 1930 (119) is still accepted:

> Silicosis is a pathological condition of the lungs due to inhalation of silicon dioxide. It can be produced experimentally in animals.

Symptoms

Inhalation of silica dust usually is not accompanied by evidences of irritation and if free from other irritating dusts may be breathed without arousing suspicion of its dangerous character (99).

The following discussion of symptoms of silicosis is based on the findings of 9,662 examinations of 7,722 employees at the Picher Clinic of the Bureau of Mines in 1928 (88).

Shortness of breath has been generally recognized as the cardinal symptom of silicosis. Dyspnea accompanies exertion in the earlier stages of silicosis and increases progressively until in the later stages it often prevents any labor. The apparent distress on exertion frequently noted in silicotics who deny shortness of breath seems out of proportion to the very slight rise in pulse rate taken 2 minutes after exertion.

Chest pains were admitted frequently, generally anteriorly, and more often on one side only. In many instances the pains seemed of a reflex character. In nearly all cases they were vague and flitting and did not seem to interfere with manual labor. Pains in the shoulder and possibly in the posterior part of the chest probably are considered by many of the men as "rheumatic"; such pains seem partly responsible for the large number of complaints of rheumatism.

Cough is a symptom frequently admitted in silicosis and generally is unproductive.

Expectoration as a symptom was admitted infrequently. When present in uncomplicated silicosis the sputum usually is clear or has a bluish tinge and is of a viscid, tenacious consistence, very difficult to cough up. The color of expectoration may be due to the color of rock mined.

Hemoptysis appeared to be more common among silicotics than was expected. In some instances, particularly in second- as well as in third-stage silicosis, fibrotic areas may mask a tuberculous lesion which remains unrecognized until it becomes an open lesion, when the sputum will show the tubercle bacilli or hemorrhage will call attention to the true condition.

It seems probable that some of the admitted night sweats were not true pathological night sweats, but the relatively high incidence in the silicotic groups appears difficult to explain except on the assumption that at the time of the sweats some active infectious process was in progress. This could be due to tuberculosis; it may have been due to a pyogenic infection or malaria, as 33 percent of the silicotics examined gave a history of previous attacks of malaria.

The loss of strength admitted by silicotic in most instances probably was connected with their dyspnea on exertion and was due to respiratory insufficiency. This probability is strengthened by the small number of silicotics with poor physical development (4 of a total of 1,062).

Gastrointestinal symptoms were pronounced in the more advanced cases, and loss of appetite was admitted. Other pronounced symptoms in advanced cases of silicosis were constipation, epigastric discomfort, and other vague complaints which the men termed "indigestion." The causes of the gastrointestinal symptoms were not ascertained.

Sayers (120) summarizes the clinical findings in silicosis as follows:

> The practically constant clinical signs are: A certain lack of elasticity of the chest wall during the movements of respiration, together with a somewhat reduced air entry, and a characteristic alteration of the inspiratory murmur from the normal "vesicular" character to a higher pitched or "harshened", "thinned", and commonly somewhat shortened type, the expiratory murmur, although somewhat prolonged, remaining fainter than the inspiratory.
>
> This type of breath sound is very characteristic, with some modifications, of silicosis in all its stages, and this clinical sign has also the significant character of more or less complete generalization. It is first noticeable at the anterior, lateral, and basal regions.
>
> In the minority of cases, however, the breath sounds may be simply diminished, but breath sounds which are simply diminished, or which, on the other hand, are merely somewhat louder or more "pronounced" than normal are not specially characteristic of silicosis.

Usually there are no accompaniments, but a stray ronchus may be heard here and there.

This complex of physical signs is almost constantly present in cases of a slight degree of "simple" silicosis. The cough may be put down to the coincident bronchitis and bronchiolitis, the recurrent pains to slight intercurrent local pleurisies.

Pathology

According to Pancoast (22) the pathological features of pneumoconiosis are (a) entrance of dust; (b) "dust cell" or macrophage; (c) entrance of the dust cell into the lymphatic system of the lungs as a carrier of silica or other particles; (d) influence of the deposited silica in the production of fibrous tissue; (e) action of silica; (f) elimination of dust; (g) predisposition of the fibrotic and silica-saturated lung to respiratory infections, especially tuberculosis.

The lung, as described by Gardner (121), is an organ developed to permit an interchange of gas between the blood and the external air. As it is in free communication with the exterior it is more or less exposed to the action of atmospheric impurities, both particulate and gaseous. Certain mechanisms are provided to protect the lungs from the accumulation of such foreign particles. The nose, through which the respiratory tract opens onto the surface of the body, is guarded by a coarse filter of hair. Behind this is a series of tortuous passages with moist walls which trap many smaller particles. In addition, the nasal cavities and the remaining portions of the upper respiratory tract, the pharynx, the trachea, and the bronchi are lined by cells covered with minute vibratory hairs, the cilia. Wavelike vibrations of these hairs tend to carry particles lodging on their surface away from the lung and back toward the surface.

Particles that succeed in passing these barriers and penetrate to the terminal air spaces of the lungs are ingested by wandering scavenger cells or phagocytes, which come from the partitions between the spaces for the purpose. These cells are capable of independent movement and tend to carry the foreign particles out of the air spaces into a special drainage system known as the lymphatics. The lymphatics are minute vessels, which drain into sedimenting basins known as lymph nodes. They are situated along the course of the vessels and bronchi and at the root of the lungs where the trachea divides into the two main bronchi. For ordinary amounts of atmospheric pollution these protective mechanisms are adequate to prevent the significant accumulation of foreign particles in the functional part of the lungs. If a person continues to work in a very dusty atmosphere for long periods his protective devices cannot cope with the situation, the mechanisms themselves are damaged, and the dust particles collect where the air should be (121).

According to Simson (122), if the first line of defense is passed and particulate matter reaches the alveoli, the cells lining the alveoli are stimulated to activity. They swell, become detached from the walls, and

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develop an active phagocytic function. The particles are phagocyted and are then carried in 1 of 2 directions; some of the phagocytes, now laden with dust, pass to terminal bronchioles, whence they may be removed in the sputum; others pass into the walls of the vestibules and terminal bronchioles. In time large numbers of dust-laden cells accumulate and cause condensation of the tissues about the entrance to the primary unit. During the period of excessive dust inhalation and possibly for a short time afterward dust cells are continually reaching and entering the areas of condensed tissue at the entrance to the primary unit. Although some cells are arriving others leave the condensed areas and enter the regional lymphatic vessels, along which they pass to the minute masses of lymphoid tissue which lie between branches of the pulmonary artery and the adjacent bronchioles, vestibules, and atria. The presence of the dust cells stimulates the lymphoid tissue and causes it to become hyperplastic. With continued arrival and accumulation of dust cells in the lymphoid masses more or less well-defined aggregations are formed. The site of an aggregation can retain only a limited number of dust cells, so that with new arrivals an overflow results. The cells comprising the overflow and those that have escaped arrest pass onward in the peribronchial and perivascular lymphatic vessels, finally to be trapped in the lymph nodes at the root of the lung. In silicosis these lymph nodes are the first sites to show fibrosis.

If inhalation of injurious dust other than silica - for example, asbestos dust - is continued over a long period or over a shorter period when the concentration of dust is great a diffuse, cellular fibrosis develops in the walls of the bronchioles, vestibules, and atria. The fibrosis tends to extend locally, involving the supporting connective tissue of the adjacent blood vessels and to some extent the walls of the alveoli in the immediate neighborhood. There is usually desquamation of the epithelial lining of bronchioles and vestibules, often without evidence of a definite exudate. In very advanced cases the fibrosis may extend to the septa, to the supporting tissues of the bronchi and larger blood vessels, and to the alveolar walls. Even in these cases fibrosis is localized to the supporting tissue about the entrance to the primary unit. It may advance to such an extent that the bronchioles become constricted, sometimes retaining their circular outline in cross-section and sometimes being reduced to mere slitlike openings. Rounded nodules of cellular character occasionally are seen, but the dense hyaline type of nodule usually is absent. Dust containing only a small percentage of silica also may produce these changes, and the liability to generalized fibrosis is greater when a low-grade type of infection (not necessarily of tuberculous origin) complicates the dust effect.

The changes described above by Simson (121) appear to him as primary lesions caused by inhalation of any injurious dust, but when the great majority of the inhaled particles is composed of or contains silica a specific and localized type of fibrosis - the silicotic islet - also develops. The first evidence of the specific and localized lesion, which Simson (121) termed the "silicotic islet", is seen in the aggregation of dust cells that probably represents the site of the lymphoid mass. A small, round area of fibroblasts appears in the center of an aggregation; on further development a central core of dense, fibrous tissue is formed. It often becomes hyaline in character and in larger and older nodules takes on a whorled arrangement. A fully formed single silicotic islet consists of a central mass of dense hyaline fibrosis arranged in whorls and surrounded by a comparatively narrow zone of cellular fibrosis in concentric laminae. Scattered through this mass, lying between connective tissue cells and fibrils, are scanty large and small round cells and a little particulate matter. A deposit of fat may be seen also in suitably stained sections, the degree varying with the age of the nodule. The early fibrotic nodule thus formed is surrounded by numerous dust cells. The growth of the silicotic islet continues by successive new arrivals of dust cells and subsequent extension of the fibrosis at the periphery of the fibrotic nodule. Apart from the individual single or composite palpable islet, noninfective silicosis may appear as a "massive" type of fibrosis. Seen microscopically, such lesions comprise numerous contiguous single and composite islets. The fibrosis is of the same character as that of the single islet; there is no evidence of breaking down. The intervening alveolar tissue is much compressed and collapsed, but there is no evidence of inflammatory infiltration or of definite matting together of the individual islets. As a rule, the larger blood vessels are not constricted.

These changes, according to Goodrich (100), make the lungs less elastic, increase their bulk, make aeration of the blood less efficient, and decrease circulation through the lungs. As a result, a more vigorous respiratory effort is necessary to supply the blood with an adequate amount of oxygen and to remove the accumulated carbon dioxide in the blood. An increase in inspiratory effect is needed to create greater negative pressure within the chest in order to draw into the less elastic lungs the same amount of air, and an increase in the normal tidal volume is needed because of the decreased areation of the damaged alveolar structures and the impaired circulation through these alveoli. As the volume of the lungs increases from the continuing deposit of silica and the increasing amount of fibrous tissue the chest gradually is held and later fixed in a position of partial inspiration, and breathing becomes more and more diaphragmatic. Even diaphragmatic breathing eventually becomes less efficient because of the usual development of a basal pleurisy which causes the inelastic lungs to adhere to the upper surface of the diaphragm. Secondary infection with bacteria may cause still greater lung damage. Chronic bronchitis or bronchiectasis may be caused by streptococcus, staphylococcus, pneumococcus, or other pyogenic organisms, as evidenced by investigations of the United States Public Health Service (123) on the development of pulmonary infections in pneumoconiosis. The results of the bacteriologic and experimental study show that -

> In general, the silicotic lung is more susceptible to bacterial infection than the average lung. This is probably due to the irritation of the respiratory tissues by the inhaled dust particles which weakens the mucous membranes and renders them susceptible to infection. The toxic influence of certain inorganic dusts upon the tissues may be a contributing factor.

The relation of tuberculosis to pneumoconiosis has been studied to a considerable extent, but comparatively little work has been done in connection with other infectious processes of the lung, e.g., pneumonia, lung abscess, bronchiectasis, and influenza. An investigation was made of these conditions, both bacteriologically and experimentally, with the view of obtaining a better understanding of the predisposition to, and the mechanism of, infection of the lung in certain dusty trades.

Bronchiectasis, lung abscess, and gangrene occur frequently in hard-rock miners. It has been definitely established that aerobic, pathogenic bacteria, and certain fungi are responsible for these conditions, but the high percentage of cases in which the anaerobic microbes of the mouth and throat have been reported would suggest that they at least participate in the etiology of the diseases.

According to Tillson (124) many of the phagocytes of the lungs, when carrying mineral particles, become trapped in the lymph channels and nodes and therefore are not eliminated. Those free in the blood stream are charged positively at the hydrogen-ion concentration of the blood and move to the cathode in an electric field. Bacteria carry a negative charge and therefore are attracted to the phagocytes, are adsorbed, and then ultimately absorbed. The agglomeration of bacteria increases the efficiency of this process, as the large phygocyte cell can dispose of many bacteria in one contact. This clumping of bacteria depends upon the plasma salts being at a high enough concentration; otherwise, bacterial absorption becomes a slow process.

Silicosis is a progressive disease. If enough silica has been inhaled to initiate nodule formation each focus continues to enlarge until a state of equilibrium is established. Nodules not visible by X-ray when a men leaves a silica industry may subsequently increase enough in size to be readily detectable some years later. The progression of the process is favored and accelerated by the development of pulmonary infection (121). In fact, many pathologists doubt whether the fibrosis of the lung that constitutes the essential pathological nature of silicosis ever arises independently of a primary infectious process (125). Rist, Mayel, and Donbrow (126), after studying the question from the medico-legal point of view, are convinced that silicosis cannot develop other than in persons already infected with the tuberculous virus and hence refuse to regard silicosis as a true occupational disease.

In some cases the infection remains latent, presents none of its usual manifestations, and merely modifies the character of the reaction to inhaled silica dust. In others it develops simulataneously with the silicotic reaction with a more or less typical localization in the upper part of the lungs. Such cases may exhibit none of the characteristic symptoms of tuberculous intoxication for many years and often are discovered only in routine roentgenographic examinations of large groups of active employees. Ultimately these men develop symptoms, expectorate tubercle bacilli, and finally die of tuberculosis, but the course of their disease is protracted. There are still others whose roentgenograms show so much evidence of tuberculosis that the characteristic features of silicosis are obscured. Nevertheless, they have long been exposed to dust, and post-mortem examination will reveal the nodular fibrosis produced by silica. Like those of the preceding group, these cases run a definitely chronic course and may not die of their infection until the fifth or sixth decade. Finally, there is another group, with well-developed nodular silicosis, whose members apparently have never had a tuberculous infection. Such men become infected, develop a rapidly progressive tuberculosis, and die within 6 months. In them the symptoms of intoxication are more acute, but bacilli may be very difficult to detect in their sputa and even in the lungs removed at autopsy (121).

Gardner claims (121) there is no difficulty in demonstrating that silica produces a reaction in the body specifically favoring multiplication of tubercle bacilli. Kettle injected a definite quantity of fine silica particles beneath the skin of one flank of a white mouse and in the opposite flank the same quantity of aluminum oxide particles. A large dose of tubercle bacilli was then injected into the tail vein of the animal. The blood distributes these bacilli quite uniformly to all parts of the body, but if the animal is killed after several days large masses of them will be found at the site of the injected silica. The bacilli are no more numerous where the alumina particles have localized than in any other part of the body. Apparently the reaction induced by the silica produces a favorable medium for the growth of these bacteria, but they disappear later and may be very hard to find. The same seems to be true of the sputum of men with silicosis and tuberculosis.

Gardner (121) reiterates that only silica and a limited number of the silicates are known to produce definite and serious pulmonary damage. In the case of silica this is associated with specific indisposition to tuberculosis and pneumonia. Of the silicates asbestos is definitely recognized as a cause of pulmonary fibrosis. Its importance in predisposing to tuberculosis is not yet settled.

At the International Conference on Silicosis (119) it was agreed that the microscopic pathological changes that may be produced by the prolonged inhalation of silica dust are:

(a) The development of a condition designated in South Africa as a dry bronchiolitis, characterized by an accumulation of dust-filled phagocytes in or in relation to the terminal bronchioles, with possibly some desquamation of their epithelium.

(b) The accumulation of dust-containing phagocytes about and in the intrapulmonary lymphoid tissue and their transportation through the lymphatics into the tracheobronchial lymph nodes. (The conditions described above under (a) and (b) do not constitute the disease silicosis.)

(c) The gradual development of fibrous tissue within such accumulations of phagocytes and the formation of characteristic nodules of hyaline fibrous tissue.

(d) Degenerative changes in these foci.

(e) The hyaline nodules increase in size by extension of their periphery. Coalescence of adjacent nodules takes place and brings about involvement of further areas of the lung. (The conditions described under (c), (d), and (e) constitute the disease silicosis.)

Macroscopically the changes observed in silicosis are:

(a) In the early stage.- A variable number of palpable pearly-white nodules up to 2 or 3 mm in diameter on the pleural surface of the lung. On section, the cut surface of the lung is studded with pigmented foci, widely scattered, a moderate proportion of which are only just palpable. The tracheobronchial lymph nodes are slightly enlarged and deeply pigmented and may exhibit foci of fibrous induration.

(b) Later stages.- The fibrotic nodules are increased in number, size, and density, and coalescence of these may be found. The portion of the lung between the fibrotic nodules may be emphysematous. The tracheobronchial lymph nodes may be smaller in size than those seen in the early stage and are fibrosed.

It was also agreed that the presence of tuberculous infection usually modifies the pathological appearance, and special attention was drawn to the following three types:

(a) In which the picture of silicosis above described may be little, if at all, modified, but in which only a biological test can demonstrate the presence of B. tuberculosis.

(b) In which the coexistence of silicosis and typical tuberculosis lesions is easily recognizable.

(c) In which the presence of tuberculosis is easily recognizable, but the existence of silicosis is more difficult to determine.

In *massive* silicosis cardiac hypertrophy and subsequent dilatation may occur. In silicosis with infective processes cardiac changes may also occur. No evidence was adduced in regard to involvement of kidney or liver.

Stages

The Committee on Pneumoconiosis and the Committee on Standard Practices in Compensation of Occupational Diseases of the American Public Health Association in a report on silicosis (127) described the stages of the disease as follows:

The disease is divided arbitrarily into first, second, and third stages for convenience of description and possible compensation purposes.

First stage (corresponds to anteprimary stage of South Africa).- The symptoms of uncomplicated first-stage silicosis are few and often indefinite. The man may apparently be quite well and his working capacity not noticeably impaired. Slight shortness of breath on exertion and some unproductive cough, often with recurrent colds, are the most usual symptoms. The man may have a little less ability to expand his chest than formerly and the elasticity of the chest may be slightly impaired. The earliest specific indication of the presence of silicosis is the radiographic appearance, consisting of generalized arborization throughout both lung fields with more or less small, discrete mottling.

This characteristic mottling is due to shadows cast by the discrete individual nodules of fibrous tissue in the lungs and is essential to the diagnosis of silicosis; without this finding the diagnosis of silicosis is not sustained except by autopsy.

Second stage (corresponds to primary stage of South Africa).- A definite shortness of breath on exertion is usually found, and pains in the chest are a frequent complaint. A dry morning cough is often present, sometimes with vomiting, and recurrent colds are more frequent. Even then the man's appearance may be healthy but he is dyspneic on exertion, he cannot work as well as formerly, his chest expansion is noticeably decreased, the movement being sluggish and diminished in elasticity.

The characteristic radiographic appearance is a generalized medium-sized mottling throughout both lung fields. The shadows of the individual nodules are for the most part discrete and well defined on a background of fibrous arborization, but there may be here and there larger but limited opacities due to irregular pleural thickening or to a localized aggregation of nodules.

Third stage (corresponds to the secondary stage of South Africa).- In the third stage the shortness of breath is marked and distressing even on slight exertion. The cough is more frequent; the expectoration is in most cases slight but may be copious. The individual's capacity for work becomes seriously and permanently impaired; his expansion is greatly decreased even with forced inspiration; he may lose flesh; his pulse rate may be increased, and his heart may become dilated.

The radiographic appearances in the third stage are further accentuated, the mottling is more intense, the nodules are larger and take on a conglomerate form so that large shadows are shown corresponding to areas of dense fibrosis.

Physical examination of an individual may reveal changes in percussion and auscultation, mild in the first stage and increasing with the progress of the disease. These alone are not sufficient to be of great value in diagnosis of silicosis.

Pancoast and Pendergrass (128) question the wisdom of designating any appearance of pneumoconiosis by any term denoting numerical stages of progress. They prefer to designate the appearance by a term which implies its pathological nature:

> Now this appearance of prominent hilum shadows and increased prominence of trunk shadows and linear markings, with or without the faint haze, has in this country at least been designated as the first stage of silicosis or pneumoconiosis. There may be some excuse for continuing to call this the first stage, but continued experience with cases of pneumoconiosis developing in various industries has led us to question the wisdom of designating any appearance of pneumoconiosis by any term denoting numerical stages of progress. * * *. We prefer to designate the appearance by a term which implies its pathological nature and to call it not a stage but the perivascular-peribronchial-lymph node type or preponderance of the condition. * * *.
>
> In cases with silicosis which is moderately slow in its progression the microscopic nodular lymphoid deposit proliferations tend to become conglomerate and to produce a macroscopic nodular process * * *. As this appearance was found as a more advanced stage of silicosis in the earlier cases examined, and especially among miners in this country, it was and is still called the second stage of the condition. It is conspicuous by its absence or insignificance, however, in many industries, notably the granite cutter, sand-blaster, sandstone and asbestos workers. It is especially likely to be absent or inconspicuous in those who are developing silicosis rapidly, such as in those working with pulverized sand without adequate protection.
>
> If this appearance is absent or nearly so as a stage of progress in so many industrial silicoses, why designate it as a numerical stage of progress at all? We have ceased to do so, and have called this appearance the nodular type or preponderance of silicotic fibrosis, or, more correctly, silicosis.
>
> There is another type of silicotic appearance which was very puzzling to us at first because of its exact identity and our inability to find the proper place for it in a numerical classification of progressive stages. * * *. Fortunately a definite pathological place seems to have been found for the condition and a roentgenological classification for the appearance. * * *. We have designated this as the interstitial type or predominence and have subdivided it into rapid and slow, which subclassification must depend largely upon the physical factors and time of exposure. * * *.
>
> This type of fibrosis will almost invariably progress to the terminal stage with little or no appreciable nodular appearance. Care must be exercised in differentiating it from a chronic interstitial lung change resulting from pneumonia or continued upper respiratory infection as in the sinuses. * * *.

The condition responsible for this appearance in advanced form and of rapid development has been termed "acute silicosis" by Chapman and some others. * * *.

There are few if any arguments to be recorded in connection with the terminal stages of silicotic fibrosis. Instead of the third stage, we have preferred to call this aspect of the disease the terminal diffuse fibrosis. It is more or less incapacitating in practically all instances, and all too often completely so.

With respect to the classification of silicotic appearances based upon known pathological changes, Pancoast and Pendergrass (128) suggest the following in place of the old numerical stages:

1. Peribronchial-perivascular-lymph node predominance or type. This may be rapid or slow, usually the latter.
2. Early interstitial predominance. This may be extremely or moderately rapid, depending on the silica intake. It may or may not have an associated slight nodular appearance.
3. Advanced interstitial predominance.
4. Nodular predominance. Rapidly or slowly progressing.
5. Advanced diffuse or terminal fibrosis.
   Conglomerate nodular type.
   Interstitial type.
   Massive fibrotic type.

Diagnosis

According to the Silicosis (Medical Arrangements) Committee of Great Britain (109) silicosis can be diagnosed even in its early stages, but the diagnosis is not free from difficulty. For instance, the clinical signs of silicosis resemble to a great extent those of other chronic diseases of the chest; further, silicosis may be complicated by the presence of tuberculosis in any stage. Irvine (129) states that it is impossible to deduce a practical standard of diagnosis from accounts of the disease furnished separately by the pathologist, the radiologist, and the clinician. Clinical examination is obviously an essential factor in the decision in any individual case, since it provides important information otherwise not obtainable regarding the general and local condition of the patient, the degree of incapacitation, if any, and the presence or absence of complication by active infection or by disease of other organs; but in a disease like silicosis clinical evidence in many cases is inconclusive and may be misleading. Other means of diagnosis in the living subject are radioscopy, radiography, and in the last resort the pathological condition found after death. Radiography also has its limitations, as on the one hand not all forms of pulmonary fibrosis revealed by it, even in miners, are of silicotic origin, and on the other hand certain types of radiograph, which do not show unequivocally "specific" signs of silicosis yet in certain circumstances may be regarded legitimately as affording evidence of the presence of a silicotic factor in the case. Accurate knowledge from which to formulate a reliable general standard of diagnosis can be obtained only by correlation of the results of pathological observation in a large number of cases with radiographs taken from the chests of the same individuals shortly before death and with the results of clinical examination made during life. The final adjudication in any individual case must always incorporate the additional evidence supplied by expert clinical examination, which indeed is frequently the deciding factor, particularly in borderland cases.

Sayers (120) includes under "clinical manifestations" those factors that can be ascertained from the individual having the disease - personal data, past history of diseases, occupation, present history, physical examination, and roentgen and laboratory observations. Levey (111) states that it is inadvisable to overlook any diagnostic medium in the early stages of silicosis. The importance of the serologic examination and the correlation of the physical examination with the past history is so great as to render all the steps of the complete examination of almost equal importance. Although it is frequently said that no diagnosis of silicosis can be made properly without roentgen-ray examination such examination must not be considered adequate, except in advanced stages. The examinations at the Picher Clinic (88) included collection of data on race, family history, personal history, past history of illness, occupational history, and physical, X-ray, and laboratory examination. A brief summary of some of the results of the examinations follows:

Family history.- The incidence of silicosis or silicosis complicated with tuberculosis was found to increase as the family histories of the men were classed as good, average, or poor.

Personal history.- The use of tea, coffee, or tobacco apparently has no relation to silicosis or tuberculosis; but after silicosis has developed, the use of alcoholic drinks and patent medicines increases decidedly; the men take these to alleviate the symptoms of silicosis so that they can continue work.

A study of age groups in published results of several investigations on silicosis shows that in the early years of the occupational life of those in dusty occupations the incidence of silicosis is low, but with increase in the age group, regardless of length of employment, it increases slightly up to about the age of 40, after which there is a marked increase. Age, therefore, has an important bearing on the occurrence of silicosis and should be considered in employing men for work in dusty atmospheres.

No correlation could be made between silicosis or tuberculosis and overcrowding in sleeping accommodations. The number of hours of sleep bore no apparent relation to silicosis or tuberculosis.

Past history of illness.- The data seem to indicate that all infectious diseases of childhood except diphtheria and scarlet fever probably decrease resistance to tuberculosis and silicosis. Picher is free from malaria, but many of the men reported they had had malaria in the past; among these men the incidence of tuberculosis and silicosis was comparatively high. Tonsillitis, especially if the attacks are recurrent, seems to be associated with tuberculosis but apparently has no relation to silicosis. The highest incidence of silicosis or tuberculosis, or both, was among men who reported bronchitis, pleurisy, and asthma. The lowest incidence was among those who reported influenza, pneumonia, coryza, and hay fever. An unknown and disturbing factor in the attempt to discover a relationship between silicosis or tuberculosis and other diseases was the difficulty in determining in many instances whether they occurred before or after silicosis and tuberculosis had been contracted. The data show that the incidence of physical defects increases among men with silicosis and tuberculosis. Several explanations are offered for this fact: Men with silicosis or tuberculosis usually are somewhat older than men in the essentially negative group; the silicotics have had a much longer period of service in the mines than the negative subjects; certain physical defects may increase susceptibility to silicosis.

Nasal obstruction and chronic catarrh, enlarged turbinates, adenoids, or other conditions that cause mouth breathing are associated with an increase in the incidence of silicosis. Persons having a vertical hanging heart are more likely to develop tuberculosis or already have it; this finding agrees with South African experience.

Occupational history.- A detailed statement of occupation is the most important single item in the history of silicotics. The mere statement that the man is a miner has slight value, if any. Many underground occupations expose the workmen to very little dust, especially in well-operated mines. The Picher histories included occupation before mining, past occupation underground, number of years in present occupation, kind of mineral mined, and total years in metal mines. All examined were divided into two groups according to duties - those exposed to large amounts of dust (those employed at the face) and those exposed to relatively small amounts (those away from the face). The data indicate that the men in certain positions contract the disease more quickly than those in other positions.

With the development of first-stage silicosis there was a drop in the number of weeks and shifts worked, and with second-stage silicosis there was a marked drop in amount of work performed on each shift. Men with silicosis and tuberculosis worked intermittently during 15 weeks of the year, while third-stage silicotics worked part of only 9 weeks; the number of shifts worked by these two groups, however, was almost the same (54 and 52).

Physical examination.- No marked symptoms of ill health, such as paleness, loss of subcutaneous fat, or decrease in muscular development, appear until silicosis reaches the third stage or until it is complicated with tuberculosis. Men with first- and second-stage silicosis appeared, on the whole, to be in better health than those in the essentially negative group.

As silicosis advances weight shows a slight tendency to increase until the third stage is reached or until the disease is complicated with tuberculosis. This increase may be due to some extent to the increase in age.

An analysis of systolic blood pressure by age groups shows that the blood pressure of the younger men did not change appreciably but that the average of the older men who had silicosis was slightly higher than for those in the corresponding age group who were essentially negative. Clinical observation shows that the blood pressure of silicotics tends to remain normal or increase only slightly, but in tuberculous cases it shows a definite tendency to drop. Any persistent marked drop in the blood pressure of silicotics indicates probable tuberculous infection.

It is generally accepted that the cardinal physical finding in silicosis is diminished chest expansion. Emphasis should be put not upon chest expansion but rather upon the importance of comparison with a man's earlier chest expansion. Probably if the vital capacity were taken on a large unselected series for comparison the decrease in vital capacity in silicosis would be greater than any chest-expansion measurements, however carefully taken, would indicate.

In the chests of miners generally, particularly those with silicosis, certain breath sounds rarely noted in the chests of healthy people not working in dusty occupations are often detected.

The slight changes revealed by examination of the heart probably could be attributed to the ages of the groups of silicotics.

Roentgenological Aspects of Silicosis

It is generally accepted that the X-ray offers the best and most reliable indication of the lung changes that occur in silicosis, particularly in the early stage. The value of this method of diagnosis depends largely upon the skill of the technician who takes the pictures and the experience of the reader who interprets them.

According to Pancoast and Pendergrass (130) the ability to interpret roentgenograms of cases of pneumoconiosis properly must be based upon several very important factors which may be enumerated as follows: A knowledge of the anatomy of the chest and many of the physiological problems associated with its anatomical constituents; thorough familiarity with normal roentgenographic and fluoroscopic appearances and permissible variations therefrom within normal limits; knowledge of the histology of the lungs and especially of the lymphatic system; clear perception of the pathology of the condition of pneumoconiosis and of all conditions which may simulate it in roentgenographic appearances; experienced intimacy with the roentgenographic appearances of the condition in question and of those that resemble it, based upon fundamental knowledge of the pathology represented; knowledge of the physical factors involved in the production of the suspected or alleged pneumoconiosis; and the employment of the proper technic to show to full advantage any or all of the abnormalities present, for technic may fully enlighten, may so modify appearances as to be confusing, or may be quite misleading.

In a discussion of the X-ray findings of the Picher Clinic (88) the fibrotic changes revealed by the study are classified arbitrarily as definitely negative, more fibrosis than usual, decidedly more fibrosis than usual, three stages of silicosis, and silicosis plus tuberculosis.

Definitely negative.- In the definitely negative chest the hilum shadows are not more than 7½ cm in width from the midline and do not cover more than 2 interspaces and 1 rib at a target distance of 48 inches. The hilum shadows occasionally may show one or more fairly large calcifications.

The pictures disclosed little if any evidence of an unusual amount of peribronchial thickening in any of the definitely negative chests. The bronchial tree usually cannot be traced farther than the inner edge of the midthird of the lung and never beyond the inner section of the outer third of the lung. The areas between the shadows thrown by the bronchial tree are clear and show no evidence of any inflammatory changes.

More fibrosis than usual.- The hilum shadows are larger than normal - that is, more than 7½ cm wide (from midline) - and cover more than 2 interspaces and 1 rib. Most of these cover more than 2 interspaces and 2 ribs. The calcifications within the hilum shadows show a very definite increase and sometimes are very large. The bronchial tree shows definite thickening and often extends well into the apexes. In the majority of cases its branches can be observed radiating to the outer third of the lung.

Decidedly more fibrosis than usual.- This class corresponds to that sometimes referred to as presilicotic. The hilum shadows are definitely enlarged and dense. The X-ray reading showed that 94.46 percent of these cases at Picher had large, dense, hilum shadows. The bronchial tree shows a definite thickening and extends to the periphery of the lung, involving the bronchioles and possibly the air vesicles.

First-stage silicosis.- The hilum shadows are increased in density and are larger than normal. In many instances (of Picher cases) large calcified spots were noted in or around the hilum shadows. These were larger than those generally observed as the result of tuberculosis in childhood. In several instances collections of such calcifications involved the entire hilum.

Following enlargement of the hilum shadows, the entire bronchial tree increases in density. It is very noticeable and often can be traced to the outer margins of the lungs. Along the thickened bronchial tree near the hilums are small shotlike spots, sometimes described as "beads" or bronchial "buds." When the spots become noticeable throughout the lower section of the lungs the case is classified as the beginning of first-stage silicosis. The spots are fairly dense, one eighth inch or less in diameter, discrete, with irregular fuzzy outline. With advance in the disease the spots increase in number, density, and size.

The diaphragm was humped in only a small number of the Picher cases in early silicosis, and no displacement of heart shadow as a result of the disease in this stage was observed.

Second-stage silicosis.- The hilum shadows are large and dense but generally do not show any more definitely than in first-stage silicosis. The beads or buds along the bronchial tree become larger, more numerous, denser, and clearer in outline; the condition is best described as a general "mottling." The mottling is bilateral, and the density is about equal on both sides, indicating that the changes started in both sides at or about the same time.

The diaphragm is often humped or "peaked", and numerous bands of adhesions are noted at the bases. The heart shadows usually are normal.

Third-stage silicosis.- The spots described under second-stage silicosis tend to coalesce, forming large areas of marked density. In some of the cases very large areas of marked density were observed - usually in the middle section of the lungs - which were similar to tuberculous consolidation; these areas, however, usually were bilateral and blended so perfectly with the snowstorm appearance as to suggest silicotic areas, which the physical examinations and laboratory findings tend to confirm.

Silicosis plus tuberculosis.- The ordinary silicotic findings described under the different stages of silicosis were noted in the Picher cases.(88). In many of them detection of the beginning of tuberculous infection was difficult because of the extensive silicotic changes. The hilum shadows were more likely to show calcified spots than in uncomplicated silicosis. In some of the cases the calcified spots or glands were very large and occasionally occurred in large numbers.

With the beginning of tuberculosis areas of marked density were observed, usually at one or both apexes. These areas were not so dense or opaque as the fibrotic areas of silicosis and appeared cottony or wooly. Usually they were unilateral in the beginning but often became bilateral before death. In some instances these areas occurred in the midsection of the lungs opposite the hilum. Large areas of marked density may occur in this region, and it was difficult to determine by X-ray whether the area was a walled-off tuberculous abscess or a dense fibrotic area of silicosis.

Pancoast and Pendergrass (130) summarize the roentgenological phases of the condition, which they had described previously, as follows:

> 1. The perivascular-peribronchial-lymph node aspect is due to the relaying of phagocyted dust to the pulmonary lymph nodes and their subsequent enlargement and ultimate partial fibrosis to the gradual enlargement of lymphoid deposits along the course of lymph vessels and the subsequent thickening of these vessels and stasis of contents. This is characterized roentgenographically by increased prominence of the hilum and trunk shadows and linear markings. This appearance is by no means characteristic of pneumoconiosis alone, and even if it does indicate the

condition, the phase is absolutely not incapacitating. It, together with a barely perceptible appearance of macroscopic nodules, corresponds to the so-called first stage.

2. The nodular aspect is due to the gradual enlargement of lymphoid deposits and their coalescence into quite apparent macroscopic nodules symmetrically scattered throughout both lungs. This corresponds to the so-called second stage. It is conspicuously absent in many industries, especially when the silica intake is rapid.

3. The interstitial type of the condition results from a hilumward and pleuralward block in the lymphatics and the escape of dust phagocytes in large numbers into the interstitial interalveolar tissue and subsequent fibrosis. It appears as a faint homogeneous haze, first on the right side, then on the left. If the silica intake is comparatively slow, it may accompany the perivascular-peribronchial-lymph node aspect, but if more rapid, it may be associated with the nodular type or may progress without the latter directly into the terminal stage of the condition without any evidence of nodulation. The unprotected or inadequately protected sand-blaster, sand pulverizer, and sandstone abrasive worker or granite cutter have been among those especially prone to present this rapid interstitial aspect.

4. Terminal and incapacitating silicosis is characterized by three general appearances - a terminal diffuse fibrosis of a conglomerate nodular type, one which is quite similar in appearance to a generalized chronic fibroid tuberculosis, and the terminal stage characterized by large fibrous consolidated areas, which closely resemble tuberculous consolidations quite frequently, but which we are now learning to differentiate one from the other.

According to Gardner (121) anatomic and roentgenologic studies of the lungs of persons exposed to various types of industrial dusts have demonstrated that all inhaled foreign substances do not produce the same kind of pathologic reaction. He divides the forms produced into three catagories - linear, nodular, or diffuse in character. A linear pattern characterizes the general type of response to most inhaled inert foreign materials; nodular lesions apparently are confined to silicosis; and diffuse reaction is exemplified in asbestosis. Mixed patterns are produced by dusts such as granite, which is composed of several different elements.

According to Haldane (131) the diagnosis of silicosis, apart from a history of serious exposure to dangerous dust, is extremely difficult, and the sooner some sort of agreement can be reached as to the grounds for a diagnosis of silicosis the better; but these grounds must be consistent with known facts regarding the infrequency or frequency of phthisis in the kind of occupation in which the patient has been actually engaged - for example, ordinary collier's work or work in driving a road through highly siliceous rock without adequate precautions.

Smith (132) has pointed out striking discrepancies between symptoms and physical signs and pulmonary fibrosis as shown by the X-ray. In a study of rock drillers in the vicinity of New York 25 percent of those whose X-rays showed unmistakable silicosis had no symptoms whatever, and in a recent study of granite workers (133) in the same locality 6 cases of 17 with advanced silicosis had no symptoms. Absence of symptoms does not indicate absence of silicosis. Smith quotes an incident reported by Watkins-Pitchford; of a group of 541 miners who had been receiving compensation for silicosis on the basis of a physical examination alone 41 percent were found not to have it when X-rays were introduced. She considers a physical examination somewhat of a luxury where large groups of workers are being examined for silicosis; where economy of time and expense is desirable it would seem legitimate to limit the examination to a good roentgenogram supplemented by occupational history.

McNally (134) states that during life the diagnosis of silicosis depends on the history, clinical examination, and roentgenographic evidence of the disease. Many times the roentgenograms are not decisive, so the diagnosis of silicosis, silicosis plus tuberculosis, or tuberculosis cannot be made with certainty. At autopsy the doubt may still linger; then a chemical examination for the quantitative determination of silicon dioxide aids in reaching the correct diagnosis.

How Fibrosis of the Lungs is Produced

According to Kettle (118) silicosis presents to the pathologist two fundamental biological problems: What is the exact process by which fibrous tissue is formed in the lungs in response to the presence of dust and how does dust influence the progress of a coincident infection? He considers that these problems lie at the very root of the matter, and even if the answers were known he doubts whether we should be in a much more favorable position to deal with the situation as it exists in industries. "But", he says, "if we cannot explain the how of the matter we may still hope to speak with some authority about the when and the where. We can discuss with profit such questions as the identification of dangerous dusts; whether they act chemically or physically; the influence of their physical state on their action; the influence of associated dusts; and the relationship of dust to infection."

For many years it was held that fibrosis of the lungs characteristic of silicosis was produced in response to the irritation caused by the hard, sharp, quartz crystals; that is to say, the dust was believed to act mechanically. Kettle (118) thinks that this point of view has some merit, that it is natural to think that hard, sharp quartz particles would wound and tear the soft tissues of the lung; it is, of course, a commonplace of pathology that foreign bodies in the tissues excite a certain amount of reaction which is followed by the production of fibrous tissue. This fibrosis, however, is never extreme and is never comparable to the extensive fibrous tissue formations which are the recognized lesions of silicosis. In point of fact, if a harmless dust is inhaled in large enough quantities some of it remains in the lung and causes mild fibrosis by the mere mechanical irritation of its presence. This fibrosis is never severe enough to interfere with the function of the lung, and in its distribution, particularly in its amount, it bears no comparison to that seen in the silicotic lung (118).

Kettle (118) considers solubility a primary factor in the harmfulness of silica, and he believes that a perfectly insoluble substance is incapable of causing pneumoconiosis. Exactly how the dissolved silica acts in the production of fibrous tissue is not known. His suggestion that it acts chemically as a cell poison has been severely criticized, probably on the grounds that since silica enters into the composition of animal and vegetable protoplasm it cannot be a protoplasmic poison. His reply to this criticism is that phosphorus enters into the composition of protoplasm, yet people have been killed by phosphorus and others have been hanged for poisoning them. Peacock (135) in 1860 made the first report of a microscopic demonstration of silica in the lungs. In 1866 Schmidt (136) reported sand in the lungs of all persons - except the new-born - ranging from 4.2 to 17.3 percent of silicon dioxide in the ash. According to Woskressensky (137) the lungs of individuals whose occupations do not expose them especially to dust inhalation contain an increasing amount of silica in direct proportion to the age. He found that from 3.5 to 33.7 percent of the ash of the lungs was silicon dioxide, and in the peribronchial lymph glands the amount was much higher, ranging from 18.3 to 55.6 percent of the ash. McNally (134) gives the silicon content of the lungs of 8 persons working in dusty atmospheres as follows:

Case	Ash, percent	SiO_2 per gram of dried tissue, mg	Occupation
44	10.78	8.6	Millstone sharpener.
503	17.14	14.0	Stonecutter.
327	12.36	2.4	Machinist.
429	14.58	3.6	Engineering draftsman.
463	19.99	4.3	Coal miner, 25 years.
411	8.84	5.0	Stone-quarryman, 9 years.
300	8.46	26.0	Granite cutter.
446	5.59	10.9	Zinc miner.

McNally (134) also states that the lungs of tuberculous subjects contain somewhat more silicon dioxide than those of normal persons; this may come from inhalation or directly from the blood stream, as the blood of tuberculous subjects contains more silicon dioxide than that of normal persons. He quotes Collis to the effect that all clinical observations have shown that a silicotic patient is particularly liable to tuberculous infection and that the silicotic process continues to advance after complete withdrawal from exposure.

Goodrich (100) thinks that dusts are more injurious as their chemical composition differs from that of the tissues of the human body.

With regard to the sericite theory, Gardner (121) states that, although it is too soon to comment authoritatively on Jones' thesis, silicotic nodules can be produced experimentally without sericite. Petrographic analysis of the silica employed for the animal experiments made in the Saranac laboratory has excluded contamination with sericite.

In his report on a comparative study of the solubility of finely divided rock dusts in water, kerosene, and alcohol Myers (138) makes the following statement regarding siliceous dust:

> It will be noted in test 1 that the process of solution (in distilled water) of the 5 mg quantity of siliceous dust was gradual throughout the 28-day period, solution being nearly complete at the end of this time; while the 50-mg weight of siliceous dust did not dissolve after the first 24 hours. It is believed that the finest particles went into solution very rapidly and that there was enough of them in 50 mg to saturate the solution in the first 24 hours. The 5-mg weight containing many larger particles and only a tenth as many of the extremely fine particles as 50 mg, went into solution slowly, and the solution was not saturated until the entire amount was in solution which took between 21 and 28 days.

Heffernan (139) summarized his theory of the action of silica on the lungs as follows:

> 1. Silicosis is a result of the local action of hydrated silica upon the pulmonary tissue. This action is of a physicochemical nature, and the speed of its development, other things being equal, depends upon the rapidity with which fresh silical hydrosol is formed and brought into contact with pulmonary tissue.
>
> 2. Substances which favor the formation of silical hydrosol from silica, when added to the silica dust, accelerate the development of silicosis - for example, the alkalis. Substances which retard or prevent the formation of hydrosol from silica, or which coagulate the hydrosol when formed, retard or prevent silicosis - for example, carbon, coal dust, clays, and probably many other substances.
>
> 3. The action of silica in producing the nodular pulmonary fibrosis which we term "silicosis" has not been paralleled so far by any non-siliceous substance.
>
> 4. Silica is a normal constituent of plant and animal cells; its presence seems essential in certain tissues, and its absence from the food of the organism disastrous.
>
> 5. The study of the role of silica in biology suggests that many of the processes of cell metabolism belong to the realm of colloidal physics.
>
> 6. Some characteristic cellular phenomena are explained most simply by regarding the living cell as a polyphase colloidal system.

Kraut (140) found that the silicic acid content of the blood of various normal persons fluctuates between 1 and 3 percent of the sulphate ash but that the individual maintains his silicic acid content with great constancy.

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The administration of easily resorbable silicic acid increases the silicic acid content of the blood many times the absorbed amount of silicic acid.

Lieb and Schadendorff (141) found the SiO_2 content of pathological lungs of silicon workers to be 0.3 to 1.46 percent if dried at 120° and 4.6 to 22.8 percent of the ashed tissue. Normal lung tissue showed values of 0.08 to 0.40 percent. The pathological findings corresponded with the analytical results.

In connection with the solubility of silica dust Haldane's (142) explanation of the large percentage of the silicate sericite which Jones found in the lungs he examined is that sericite is less soluble than free silica in the alkaline liquids of the lungs. Kettle mentioned (142) in this connection that crystalline silica when inoculated in the veins produces lesions but that amorphous silica does not. He also said that readily soluble silica and insoluble silica do not produce lesions but that between these two is a silica that "turns the trick." In 1929 Mavrogordato (143) gave as the cause of simple silicosis the reaction of the reticulo-endothelial system of the organ concerned to invasion by free silica, but dust phthisis, the clinico-pathological entity met in practice is superficially a variable disease. Beyond the external factors and the reticulo-endothelial system as an apparatus lies the man. Individuals vary in their ability to resist the dust they inhale. The amount of dust that can be recovered from a lung need bear but little relation to the extent or character of the lesions found.

It was agreed at the International Conference on Silicosis in 1930 (119) that:

> To produce the pathological condition, silica must reach the lungs:
>
> (a) In a chemically uncombined condition, although the dust inhaled may be either a natural mixture of silicon dioxide with other dusts, such as occurs in granite, or an artificial mixture, such as scouring powder.
>
> (b) In fine particles of the order of less than 10 microns. There is no evidence as to the lowest limit of size in which the particles may be capable of producing the disease.
>
> (c) In sufficient amount, and over a certain period of time; these two factors are reciprocal variants. The minimum of these two respective factors has not yet been determined.
>
> Silica dust plays the dominant rôle in the production of silicosis, admixture of other dusts tending to modify the picture in the direction of that of other pneumoconioses, in some relation to the proportion of free silica inhaled.
>
> There is experimental evidence that the solubility of silica in the tissues is an essential factor in the causation of silicosis.

According to Badham's summary (144) of the information supplied by various members of the International Silicosis Conference silicosis becomes noticeable after widely differing periods of exposure to siliceous dust, depending apparently upon:

1. The amount of dust inhaled.
2. The percentage of free silica contained therein.
3. The size-frequency (or fineness) of the particles inhaled.
4. The nature and sort of such other substances (including vapors and gases) as may be inhaled simultaneously or otherwise.
5. The powers of resistance of the individual concerned.
6. The presence or absence of a complication by an infective process.

In a discussion of the sericite theory as a cause of silicosis Irvine (145) stated that although it quite probably will become necessary to revise and widen the definition of the essential factors in the causation of silicosis, even so nothing in all this has significantly altered the general conception of silicosis as a definite pathological entity, which is readily identificable by the pathologist after death and which is adequately distinguishable during life by expert clinical and radiological examination, or the knowledge that this disease year by year produces many cases of incapacity or death. The position, he says, in these respects is not significantly affected by the present lack of complete knowledge regarding the exact constituents of a siliceous dust that may cause silicosis. In spite of considerable opposition from certain quarters, this view was accepted at the meeting of the International Labor Conference at Geneva, in June 1934, and the conference accordingly affirmed the international recognition of silicosis as an occupational disease and described it as "silicosis, with or without pulmonary tuberculosis, provided that silicosis is an essential factor in causing the resultant incapacity or death."

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